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TRAUMATIC RETINAL ANGIOPATHY

ARTHUR J. BEDELL, M.D.

ALBANY, N. Y.

At the 1910 meeting of the Heidelberg Ophthalmological Congress Purtscher described a peculiar condition of the fundi, consisting of exudate and superficial hemorrhages in one eye and a large preretinal clot in the other. The condition followed a head injury. Purtscher called it "angiopathic retinae traumatica" and stated that it was caused by cerebrospinal fluid in the perivascular lymph spaces of the retina and in the choroidal space. Since that time several cases have been reported. In all probability Jacobi was the first to record the white peripapillary patches and the retinal hemorrhages seen after a fracture of the skull. His description of the fundus was brief. The appended bibliography covers the majority of the cases in which the condition can be likened to or mistaken for traumatic retinal angiopathy.

Extravasation of blood in the fundus following a head injury or compression of the thorax had been described long before Purtscher's article appeared.

During the past twenty-eight years a variety of changes in the fundus have been seen as a result of trauma. No more than 40 authentic cases of traumatic retinal angiopathy are on record, and many of the citations are so brief as to be mere summaries. A few cases have been carefully observed and the clinical details reported in full. Some of these reports are illustrated by colored drawings and others by rough sketches, but only recently has any attempt been made to portray in chronologic order the changes that take place in the fundus.

Vogt and Knüsel illustrated the progress in their cases by several drawings. Chou and Danis each presented two sketches of the fundi of their patients. Recently a few photographs, including those by Stokes and by Giqueaux, have been published, and these add much to the exactness of the clinical descriptions, but, so far as it is known, neither a colored photograph nor a black and white photographic series has ever been published.

Amsler in 1934 found 25 cases reported in the literature and added reports of 5 new cases with four drawings in color. It is interesting to

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note that this observer did not refer to any cases from the United States, although several had been reported here.

REPORT OF CASES

Three new cases are recorded. The first was one of traumatic retinal angiopathy following fracture of the lumbar vertebrae and questionable head injury.

CASE 1.—D. H., a man aged 21, was seen eight days after an automobile accident. He was thrown head first through a windshield, and the third and fourth lumbar vertebrae were fractured. There were no abrasions or contusions about the head.

On the right side there were marked subconjunctival hemorrhages and ecchymoses of the lids. The pupil of the right eye was 4 mm. in diameter, regular and active. The media were clear. The entire posterior pole consisted of an immense, milky-white swelling which obscured the disk and covered the retinal vessels, resembling a collection of large cumulous clouds in the bright blue sky on a hot summer afternoon. There were many superficial hemorrhages; some consisted of striate thin streaks and others of thick red masses, most marked toward the nasal side of the disk. The darker ones were superior to the macula. Where the vessels were visible between the clouds the arteries were of normal size, but the veins were at least twice their usual width.

The left eye presented no ecchymoses or conjunctival hemorrhages. The edema of the fundus in this eye was similar to that found in the right eye, but less in extent and less thick. The retinal hemorrhages and vessels resembled those in the other eye.

Within one week both disks were clearly outlined, except in a few places where thin clouds covered the margins. The vessels were of nearly normal size.

The patient's improvement was steady; the white clouds became smaller, and no fresh hemorrhages appeared.

Photographs were taken three months after the accident. The right eye was normal externally, and the vision was 6/6. The round disk was slightly pale but clearly outlined, for only a few translucent granular white flecks lay over the edge. Remnants of similar clouds were close to the nasal side of the disk, and in a broad curved zone between the major temporal vessels. Some discrete dots were dispersed over the nerve fibers, with no regularity in position or distribution. The veins were proportionately larger than the arteries, and a few rounded hemorrhages were scattered about.

The left eye was congenitally amblyopic, with convergence of 15 degrees. The exudate, although more brilliant, more granular and denser than that in the right eye, was distributed over a smaller portion of the fundus. The arteries were narrower than those in the right retina, and several minute, round and striate hemorrhages were present. In neither eye was the field of vision abnormal.

Traumatic compression of the chest, as reported by Perthes, Béal, Stoewer, Berrisford, Schneider, Stokes and others, has produced distinctive changes in the fundus. The variations have been in the amount of blood and in the size of the fluffy clouds.

In a personal communication Stokes described a case in which a photograph of the fundus showed much blood with little white splotching. The condition in the second case is of this type.

CASE 2.[1]—C. V. M., aged 47, was injured when a motor truck overturned, pinning him beneath it. Dr. A. H. Traver, who saw him about thirty minutes after the accident, found that his face, neck and the upper part of his chest were black and that there was no perception of light. I saw the patient a few hours later.

Both eyes protruded, and there were immense subconjunctival hemorrhages. The pupils were 2 mm. in diameter, regular and active. The media were clear. There were no intraocular extravasations, but several white, radiating, cloudlike plaques were seen near the normal appearing disks.

Four days later the white areas had become much less dense and smaller, but several linear hemorrhages had formed along the major vessels, particularly toward the temporal side of the disks.

Three months after the injury the white spots and hemorrhages were still present in the right eye, the vision of which was 6/6, and the visual field was concentrically contracted about 10 degrees.

The vision of the left eye was 6/60. The hemorrhages and exudates had disappeared, but the clearly outlined disk was pale, and the arteries were smaller than before. The field of vision showed concentric contraction to 15 degrees and a central scotoma of 8 degrees.

Unfortunately, no photographs could be taken, for at that time no camera for clinical use had been perfected.

Fifteen years after the injury the patient was physically fit. The vision of the right eye was 6/6, and the fundus was normal. Vision in the left eye was 6/30 and the pupil was 3.5 mm. in diameter, regular and active. The media were clear; the oval disk was distinctly pale; the arteries were narrow, and the veins were about the normal size. The visual field was contracted to 40 degrees, with a small oval central scotoma 6 by 8 degrees.

In this case of compression of the chest, traumatic asphyxia and fracture of the transverse processes of the seventh, eighth and ninth dorsal vertebrae there was immediate blindness, with exophthalmos and subconjunctival hemorrhages. The end result was unilateral atrophy of the optic nerve.

There have been several excellent summaries of traumatic retinal angiopathy following head injury. Those worthy of special mention are the papers by Eales, Beaudoux, Stähli, Vogt and Knüsel, Best, Vogt, Merigot de Treigny, Nitsch, Rubel, Chou, Rados, Giani, Danis and Amsler. The condition in the third case is representative of this type.

CASE 3.—A man, aged 33, fell 14 feet (4 meters) from a roof, striking his head on the right side of the region of the vertex. There was a superficial laceration of the skin, which was closed with three clips. Immediately after the accident the patient noticed that he could not see with his right eye.

Nine days later, on April 16, 1937, he was examined by me. Vision in the right eye was limited to perception of light. The pupil was 3 mm. in diameter, regular and active to light stimulus. The media were clear. The disk was almost round and pale pink. The retinal vessels were of normal size and contour, with two major subdivisions. Surrounding the disk were many white, soft, fluffy exudates. Some of these were small and thin, whereas others were thick clouds through

1. Dr. Traver gave me permission to report this case.

which the underlying vessels and retina could not be seen. They overshadowed the major vessels, were thickest between the temporal branches and were at least 2 diopters higher than the retina in the region of the macula and the disk. These great billowy masses were all anterior to the vessels and resembled soft fleecy clouds, with partially clear spaces between them. There were a few striate hemorrhages paralleling the vessels and several small, irregularly round ones on the surface of the retina. Toward the temporal side of the macula there was a large, oval, preretinal hemorrhage, 3 by 2 disk diameters in size, the outer margin of which merged into a red granular wall. The dark red clot was almond shaped and 2½ by 1⅓ disk diameters in size, as measured on the film, with the broader end near the clear macula.

In the left eye vision was 6/5. The pupil was 3 mm. in diameter, regular and active. The media were clear. The disk was distinct, and the fundus was normal.

A week later, sixteen days after the injury, a great change had taken place in the right eye. The exudate was much flatter, and although it was still anterior to the level of the vessels, in only a few places was it dense enough to conceal the vessels. Several of the early hemorrhages had been absorbed, but many fresh bright red ones were scattered about the posterior pole in the region of the primary cloud. Some were on the retina, others in the exudate and still others on the surface of the exudate. The disk was a little less pink than it was a week before. The vessels were unchanged. The preretinal hemorrhage was smaller, and the encircling blood cell wall was much more attenuated.

Twenty-three days after the injury the disk was definitely pale, and the arteries were smaller. The exudate was much thinner, and only a few clouds veiled the vessels. There were several long, narrow hemorrhages overlying the vessels, particularly the two major nasal branches. Most of the hemorrhages were, however, smaller, and the only new ones consisted of fine punctate hemorrhages near the macula. Three almost horizontal wrinkles extended across the macular region. The preretinal clot measured 12 by 9 mm., with a faint outlining arc.

Thirty days after the injury the vision was 6/120. The disk was paler, and the arteries were small, with an increased central light reflex. The exudate was thinner and less extensive; the largest mass was between the macula and the disk. Several hemorrhages had disappeared entirely. New ones were present about the nasal side of the macular area, and the older ones above and below the disk were larger than they were a week before. The general retinal edema was almost gone, and the wrinkles were fainter. The macula was a little redder and more granular. A pallid halo surrounded the outer edge of the preretinal hemorrhage.

Thirty-seven days after the injury the disk was pale pink. The clouds of exudate were confined to the region between the disk and the macula; most of them were extremely thin, and only a few thick ones remained. All the hemorrhages were smaller; those along the nasal vessels were scarcely discernible. The only recent extravasation was on the surface of the exudate, near the temporal margin of the disk. The macula was red, with a dark outlining ring consisting of fine yellow dots on the side toward the disk. The clot measured 10 by 8 mm.

Forty-four days after the injury the vision was 6/21. The only marked changes were the attenuation and partial absorption of the cloudy exudate and the decrease in the size of the hemorrhages, most of which appeared as fine, flat red spots. The macula was almost surrounded by a delicate granular pinpoint layer. The hemorrhage measured 9.5 by 7.5 mm.

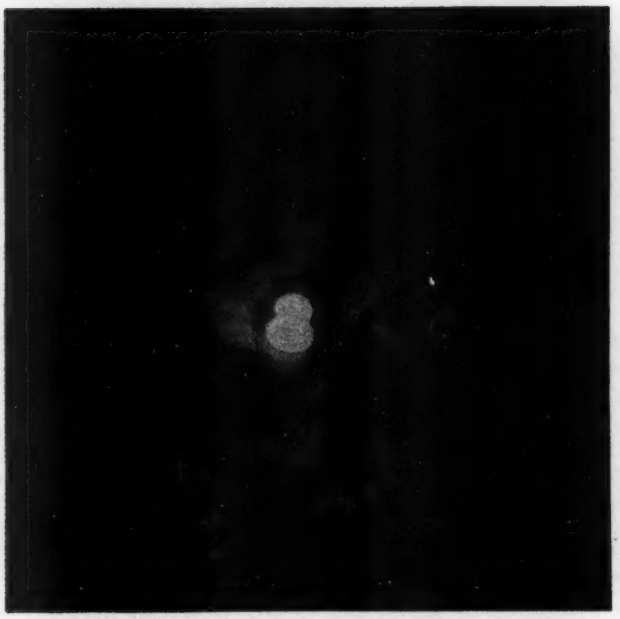

Fig. 1 (case 3).—Sixteen days after injury. There are large, soft white, fluffy clouds anterior to the retina and many hemorrhages on the retina, on the exudate and paralleling the vessels, particularly the inferior nasal vein.

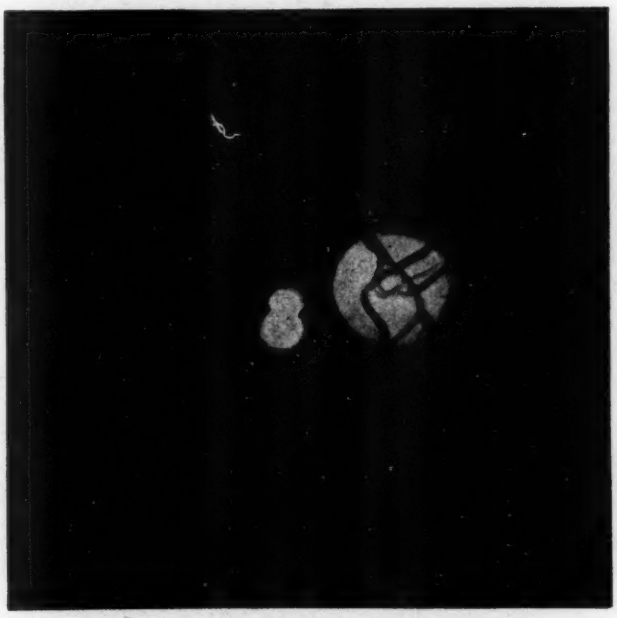

Fig. 2 (case 3).—Terminal stage, two hundred and forty-eight days after injury, showing atrophy of the optic nerve. Vision was 6/120. The disk is white, and the arteries and veins are smaller than normal. Widely distributed pale streaks are seen in the retina.

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Fifty-one days after the injury the disk was white, the arteries were much narrower, and the exudate was broken into a white film with innumerable white nodes. There were a few fresh hemorrhages; the old ones were little changed. The clot measured 9 by 6 mm.

Fifty-eight days after the injury the exudate resembled a web of delicate white spangles. There were no recent hemorrhages. The preretinal hemorrhage measured 8 by 5.5 mm.

Sixty-six days after the injury the bright silvery grains of exudate were much less conspicuous than they were a week before. The hemorrhages were still present.

Seventy-three days after the injury several white specks remained. The hemorrhages were minute, and the veins and arteries were smaller. The preretinal hemorrhage had disappeared.

Eighty days after the injury a few white dots and deep granular blood remnants were still present.

One hundred and twenty days after the injury the entire fundus was darker than it had been previously. The white nerve head, with its atrophic cup, was more distinct, and the small arteries contrasted with the dark veins.

The last photograph was taken two hundred and forty-eight days after the accident. The vision was then 6/120. The disk was white, the arteries and veins were small, and the fundus was mottled with many pale lines over the arcuately distributed nerve fibers.

DIFFERENTIAL DIAGNOSIS

The cloudy swelling of the retina that occurs in sudden closure of the central retinal artery is more or less uniform and probably always extends beyond the macula. The retina never presents the fluffy white clouds one sees in traumatic retinal angiopathy. Furthermore, the degeneration of the wall of the vessel is usually patchy, and as the white plaques are absent in traumatic retinal angiopathy, even the terminal stages are too distinct to perplex the experienced worker.

In partial closure of the artery the edema is limited to the region supplied by the branch. Hemorrhages are seldom if ever present and never occur in recurring crops. In the presence of a cilioretinal vessel the contrast between the affected and the unaffected portion of the retina is so striking that there should be no confusion or delay in making a correct diagnosis.

In endarteritis of the central retinal artery hemorrhages are frequently found in the gray retina, but no cumulous clouds are present.

Traumatic, or Berlin's, edema does not produce the pathognomonic fluffy cloud of exudate, and no recurring hemorrhages are present; the swollen retina is a uniform, gray-white layer and not uneven, like hillocks of snow. In traumatic angiopathy the vision may be lost temporarily at the time of the injury. Usually, however, the tendency is for it to undergo a progressive decrease. The reverse is true of Berlin's edema, of which poor sight is always the first and major symptom, with later recovery of all or most of the vision.

Traumatic retinochoroiditis is easily differentiated: The retinal swelling may be great and extensive or limited to a sector of the fundus. The primary edema is in the form of a gray sheet and not like billowy white clouds. The characteristic terminal spotty pigmentation eventually completes the differentiation.

The "snowbanks" of hypertensive retinal disease are rarely as rounded or as white and probably never as thick as are those of traumatic angiopathy. The showers of small hemorrhages are seldom, if ever, associated with hyperpiesia. The history of the illness and the physical examination of the patient are sufficient to remove any doubt regarding the correct diagnosis.

ETIOLOGY

The cause of this unique change is not known. The theory accepted by some is that an increased tension of the cerebrospinal fluid produces stasis about the disk. Other observers believe the change to be due to a contusion of the eyeball and consider that the fleecy white patches resemble the edema of Berlin, whereas still others believe it to be a combination of Berlin's edema and diapedesis.

Best stated that he could not conceive of liquid displacement far into the retina, as he believed that this would rupture both the vessels and the tissues.

The advocates of a fat embolus are few, but Urbanek presented a strong case in favor of his theory.

According to Vogt, the cloud is in the membrana limitans interna. This is highly probable, although a study of fundus reflexes does not preclude the possibility of these clouds forming between the retina and the vitreous or in the posterior portion of the vitreous. In some photographs reflexes are frequently seen about the disk, extending along the temporal vessels. A comparison of these with the drawings that appear in the Chou paper and a critical inspection of the photographs in case 3 would lead one to the conjecture that the lesion may be in or about the vitreous and not in the retina.

Innumerable questions crowd for answers. Just how does this massive white cloud develop? Since no pathologic examination has been made, the problem is unsolved. If sudden intracranial pressure is the causative factor, why is traumatic retinal angiopathy so rare and why does it not appear when there is an acute increase in intracranial pressure, as in hypertension or in nephritis?

When does the cloud form and how long does it last? The cloud has been seen within a few hours (as in case 2) and has lasted for many months (case 3 and others). When Mitchell first saw his patient, two months after the accident, he was impressed by the immense area of exudate in the center of the fundus. In Stokes's photographs of his patient the hemorrhages almost covered the exudate. The appearance in every case is different.

When the reports of cases of Berlin's edema, fracture of the skull and compressions of the chest are carefully analyzed, it becomes apparent that many clinicians have seen retinal hemorrhages and that in a few instances white clouds have been perceived. The hemorrhages may have overshadowed the clouds, or the observer may have failed to appraise their peculiar significance or may have regarded them as too trivial for an elaborate description or an attempted explanation.

Some new conceptions must result from an examination of the photographs in the reported cases. Among them are two of more than passing interest; first, that the exudate often precedes the hemorrhages, and second, that there is no evident relation between the amount of exudate and the visual end result.

SUMMARY

Three previously unreported cases of traumatic retinal angiopathy are here recorded. They represent the three types: (1) retinal angiopathy due to fracture of the lumbar vertebrae; (2) that due to compression of the chest and traumatic asphyxia, with fracture of the dorsal vertebrae, and (3) that due to a direct injury to the head. In the first 2 cases the lesions were bilateral, although they were less destructive in one eye than in the other. In the third case the damage was limited to the eye on the side of the injury. In the case in which the lumbar vertebrae were fractured the fundus showed the largest clouds, but the end result was the best, so that one concludes that the size and thickness of the clouds are not factors in determining the degree of permanent disability. The amount of damage to the optic nerve cannot be evaluated until the white areas begin to clear. When compression of the nerve produces a pallor of the disk, progressive optic atrophy is to be suspected, which, if present, usually ends in blindness.

The fundus picture is distinctive, and although some investigators confuse it with other conditions, nothing identical has been described. This photographic record with color prints should definitely place traumatic retinal angiopathy where it can easily be diagnosed. It is probable that many cases of mild traumatic retinal angiopathy have not been recognized or have even been overlooked entirely.

The appalling harvest of the dead and the maimed which has followed in the wake of the grim reapers—automobile speed demons and irresponsible drivers—has materially changed the professional outlook concerning head injuries.

Every person with suspected trauma to the skull should be examined by a competent ophthalmologist, for the preservation of life may depend on a correct understanding and appreciation of the fundus changes that may be present and on the institution of approved therapeutic and surgical measures for relief.

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SENSITIZATION OF RABBITS TO UVEAL TISSUE BY THE SYNERGIC ACTION OF STAPHYLOTOXIN

HUGO LUCIC, M.D.
BALTIMORE

Approximately thirty years have elapsed since Elschnig [1] first proposed the anaphylactic theory of sympathetic ophthalmia. During this time a considerable amount of evidence has accumulated, some of which indicates that allergy to uveal pigment may play some as yet undetermined part in the pathogenesis of this disease. Elschnig's work has been strongly supported abroad by several workers and in the United States notably by Woods,[2] who has elaborated on the theory.

In the main, the evidence presented in support of Elschnig's theory consists in the demonstration that uveal pigment possesses antigenic properties in the host; that it is organ specific, and that it materially lacks species specificity. The demonstration of these properties was attempted by various methods, among which the complement fixation reaction and the intracutaneous pigment test are of most value. Of these two, the intracutaneous test is easier to perform and interpret. This test was originally proposed by Woods,[3] who has used it as a diagnostic test in cases of sympathetic ophthalmia for a number of years. The positive reaction is characterized clinically by an area of redness and some induration at the site of the injection within twenty-four or forty-eight hours. The clinical interpretation of this test is by no means easy; occasionally the reaction is not striking, or a moderately severe reaction may be due to sensitivity to other beef protein or to some unknown factor. Recognizing these difficulties, Friedenwald [4] conceived the idea of examining the reaction histologically. He discovered that a

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

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3. Woods, A. C.: Sympathetic Ophthalmia: The Use of Uveal Pigment in Diagnosis and Treatment, Tr. Ophth. Soc. U. Kingdom (pt. 1) **45**:208, 1925.

4. Friedenwald, J. S.: Notes on the Allergy Theory of Sympathetic Ophthalmia, Am. J. Ophth. **17**:1008, 1934.

positive reaction was characterized by typical histologic changes at the end of two weeks which were almost identical with the histologic picture observed in eyes affected with sympathetic ophthalmia. Because of its comparative reliability, this test is now used routinely at the Wilmer Ophthalmological Institute as the final criterion of sensitivity to uveal pigment.

Woods recognized some years ago that allergy to uveal pigment is not the only factor involved in the pathogenesis of sympathetic ophthalmia. He expressed the belief that a hypersensitivity to pigment developing after a wound or trauma to the uveal tract set the stage for the development of sympathetic ophthalmia and determined the characteristic histologic picture of the disease. He also expressed the opinion that some other unknown factor, possibly nonspecific, ignited the focus after the allergy to pigment developed. The accumulated evidence of allergy to pigment, being an almost constant finding in cases of sympathetic ophthalmia, strongly supports this view.

In the experimental approach to this problem, the production and demonstration of hypersensitivity to uveal pigment are manifestly necessary before the relation of pigment sensitivity to sympathetic ophthalmia can be evaluated. Up to the present time no one has been able to produce in the laboratory animal a hypersensitivity to uveal pigment comparable to the reaction that occurs in man. Since the final criterion of pigment sensitivity in man is based on the histologic characteristics of excised skin, it is proper that sensitivity in the animal be finally judged on this basis also. After successful sensitization of laboratory animals, various methods of inciting sympathetic ophthalmia may be attempted. The purpose of this report is to show that a certain number of rabbits can be sensitized with uveal tissue and that the reaction can be shown histologically to be similar to that found in man, and to compare the reaction that occurs in the eyes of sensitized and nonsensitized animals on intraocular injections of uveal pigment.

In 1933 Burky [5] isolated a strain (Ha) of Staphylococcus which produced an exotoxin, lethal for adult rabbits. He discovered that by repeated intracutaneous injections of the toxin into a rabbit there developed not only an active immunity but a hypersensitive state to the broth in which the toxin had been produced. From this primary observation Burky reasoned that other substances with mild antigenic properties may become activated if combined with the toxin. To prove this he combined the toxin with various substances, all of which had mild antigenic properties for the rabbit, and succeeded in sensitizing this animal to such material as low ragweed extract, lens protein and homologous

5. Burky, E. L.: The Production in the Rabbit of Hypersensitive Reactions to Lens, Rabbit Muscle and Low Ragweed Extracts by the Action of Staphylococcus Toxin, J. Allergy **5**:466, 1934.

muscle tissue. These studies have been verified and elaborated on by Swift and Schultz [6] and by others. Following these observations, it seemed possible that rabbits could be sensitized to uveal tissue by making use of synergic action of staphylotoxin.

MATERIALS AND METHODS

Preparation of Uveal Tissue Toxin.—The "uveal tissue toxin" was prepared as follows: fresh bovine eyes, cleanly trimmed of all extraocular tissues, were immersed in boiling water for thirty seconds. The uvea of each eye was removed under aseptic precautions. The cornea was cut away by a circular incision at the limbus, and the eye was incised from the limbus to the optic nerve. The incision was continued around the nerve, which was removed. The eye was then everted on the forefinger, the lens and the vitreous were removed, and the retina was carefully scraped away from the choroid. The uveal tract was next removed with a pair of forceps and dropped into a large test tube containing 20 cc. of hormone bouillon. The test tubes were incubated for one week, and all the contaminated ones were discarded. The sterile tubes were inoculated with a twenty-four hour broth culture of Staphylococcus aureus, strain Ha, and incubated for two weeks at 37° C. The tubes were shaken slightly each day to aid in the digestion of the tissue. After the given interval the contents of the tubes were pooled, sufficient tricresol was added to make a 0.5 per cent solution, and the flask was left in the incubator for twenty-four hours. The entire mass was thoroughly macerated in a mortar with a little glass and filtered through sterile gauze. The filtrate was collected in a flask containing glass beads and was placed in a shaking machine for twenty-four hours. This preparation was then tested for its toxicity. An intravenous injection of 0.1 cc. per kilogram of body weight killed a healthy adult rabbit within forty-eight hours. Rabbit uveal tissue toxin was similarly prepared, except that a proportionally smaller amount of hormone bouillon was used. Material used for injections in some of the control animals was similarly prepared, except that Bacillus subtilis was substituted for Staphylococcus. This was designated as uveal tissue subtilis material.

Experimental Procedure.—Fifty-three dark, adult rabbits were used in the experiment. Twenty-two of these were used for sensitizing inoculations with bovine uveal tissue toxin and 11 for sensitizing inoculations with rabbit uveal tissue toxin. Twenty animals served as controls; 10 received similar inoculations with uveal tissue subtilis material, and 10 were similarly given injections of stock uveal pigment. Each rabbit was clipped closely with an electric clipper for intracutaneous injections. Preliminary to the sensitizing inoculations, the animals were all found insensitive to uveal pigment. They were tested with an intracutaneous injection of a stock suspension of uveal pigment, and the tissue into which the injection was made was removed two weeks later for histologic examination. Sensitizing inoculations were given intracutaneously at intervals of one week until four injections were completed. The amount of the respective preparation used for each injection was 0.1 cc., except for the initial injection, for which the amount was reduced to 0.05 cc. on account of the severe toxicity of the toxin. All the

6. Swift, H. F., and Schultz, M. P.: Studies in Synergy: The Synergic Action of Staphylotoxin and Beef Lens Extract in Rabbits, J. Exper. Med. **63**:703, 1936; Studies in Synergy: The Synergic Stimulating Effect of Hypersensitivity to Foreign Protein and to Bacteria, ibid. **63**:725, 1936.

animals were retested with stock suspensions of uveal pigment twenty-eight days after the first sensitizing injection. Tissue from the injected area was removed two weeks later for sectioning. Rabbits which received inoculations of bovine uveal tissue toxin were tested with swine or rabbit uveal pigment; those which received rabbit uveal tissue toxin were tested with swine uveal pigment; the control animals were tested with swine, rabbit or autogenous uveal pigment. The degree of the cutaneous reaction observed at the end of twenty-four hours was recorded clinically as positive, questionable or negative. The histologic reactions were recorded as positive, negative or nonspecific.

RESULTS

In 6 of the 22 rabbits which received inoculations of bovine uveal tissue toxin a hypersensitivity to pigment developed as determined by the histologic reaction. None of the 11 animals which received injections of rabbit uveal tissue toxin showed a positive histologic reaction at the end of the sensitizing period. None of the 20 control animals showed a positive reaction. The clinical reactions observed at the site of the inoculation were unreliable in these rabbits; they generally did not agree with the histologic reaction. Since the characteristic histologic reaction is comparatively more reliable, the reactions are reported on this basis alone.

To determine the reliability of the positive histologic reaction, 2 of the animals with such reactions were tested simultaneously with rabbit, swine and autogenous [7] uveal pigment. Both of the animals showed a uniformly positive histologic reaction to all three suspensions of pigment, thereby demonstrating once more the organ-specific antigenicity of uveal pigment. To determine the approximate duration of hypersensitivity, 1 animal was retested eight months later and was found to be hypersensitive at the end of this period.

HISTOLOGIC PICTURE

The histologic picture of sections taken from normal animals (figs. 1 and 2) was different from that of sections taken from sensitized animals (figs. 3, 4, 5 and 6). The former showed large masses of unphagocytosed pigment lying free in the otherwise normal tissues of the dermis. A few clumps of pigment were found within a number of monocytes. Occasionally irregular areas of mild lymphocytic infiltration surrounded these clumps of pigment, but most often no cellular reaction was seen; there was no perivascular infiltration, and the entire picture was remarkable for its absence of cellular reaction. Sections from sensitized animals showed an intense cellular reaction made up mainly of lymphocytes, epithelioid cells and giant cells. Some of the sections

7. A large piece of iris tissue was obtained from the same animal; this was ground up in a sterile mortar and suspended in a small amount of physiologic solution of sodium chloride.

showed a few polymorphonuclear leukocytes. The pigment was broken up into small particles, and it was almost completely phagocytosed by epithelioid and giant cells. Definite nests of epithelioid cells surrounded by small mononuclears were seen throughout the area containing pigment. Perivascular infiltration was seen throughout the section. This histologic picture was essentially the same whether rabbit, swine or autogenous pigment was used, and it was similar to the picture observed

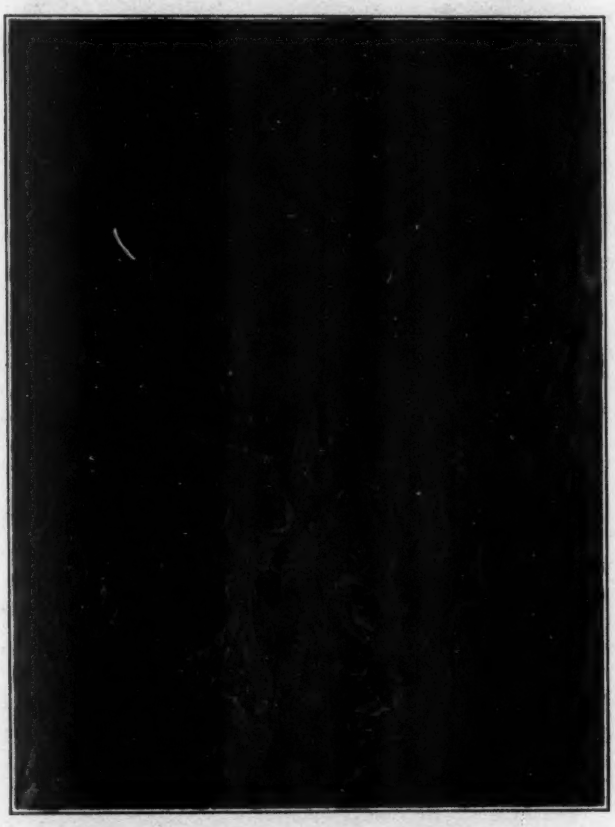

Fig. 1.—Negative cutaneous reaction for uveal pigment in a rabbit, showing comparative absence of cellular reaction. (Low power.)

in "positive" sections taken from skin of persons with sympathetic ophthalmia, as described by Friedenwald (figs. 7 and 8). The "nonspecific" reaction which occurred at the site of the inoculation of bovine uveal pigment suspension consisted mainly of a dense infiltration of degenerated polymorphonuclear leukocytes with areas of necrosis; the pigment was broken up into small particles and was scattered throughout the area of cellular infiltration (figs. 9 and 10).

Fig. 2.—High power magnification of section in figure 1.

Fig. 3.—Positive cutaneous reaction for uveal pigment in a rabbit, showing phagocytosis of pigment by giant and epithelioid cells. (Low power.)

Fig. 4.—High power magnification of section in figure 3.

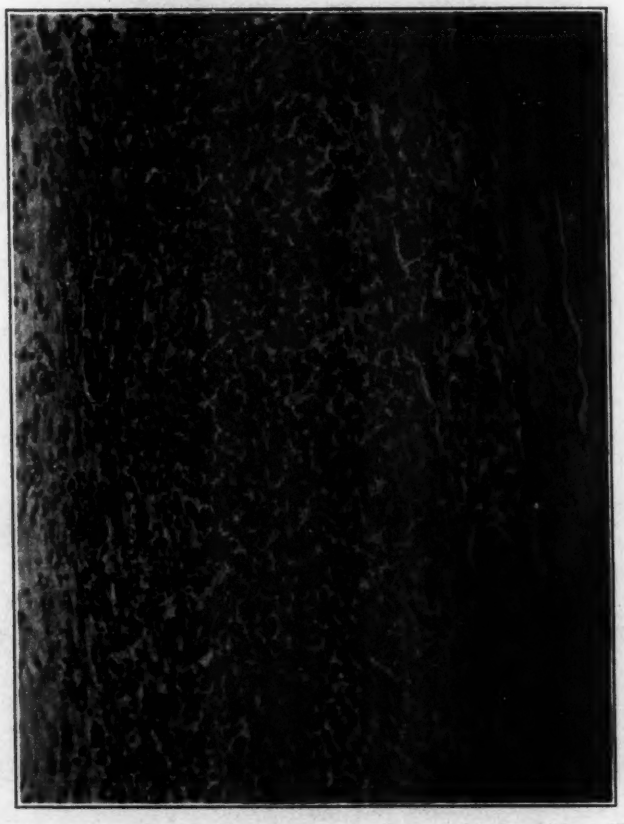

Fig. 5.—Positive cutaneous reaction for uveal pigment in a rabbit. (Low power.)

Fig. 6.—High power magnification of the section in figure 5.

Fig. 7.—Negative cutaneous reaction for uveal pigment in a human being. (High power.)

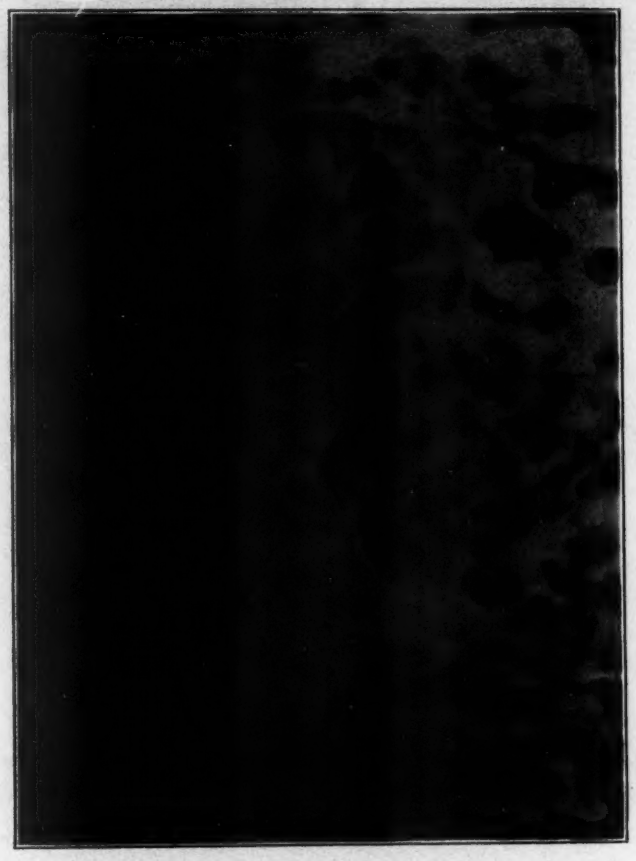

Fig. 8.—Positive cutaneous reaction for uveal pigment in a human being. (High power.)

Fig. 9.—Nonspecific cutaneous reaction for uveal pigment in a rabbit, showing infiltration of polymorphonuclear cells and areas of necrosis. (Low power.)

Fig. 10.—High power magnification of section in figure 9.

Fig. 11.—Reaction to uveal pigment injected into the ciliary body of a hypersensitive rabbit, showing partial phagocytosis by monocytes. (High power.)

INTRAOCULAR INJECTION OF UVEAL PIGMENT

Three of the hypersensitive animals were used in this experiment. One rabbit (L4) was given an injection of 0.1 cc. of a 0.5 per cent stock suspension of swine uveal pigment into the anterior chamber of one eye (left eye), and a ragged iridectomy was performed in the other eye. The second rabbit (L6) was given an injection of the same pigment into the anterior chamber and ciliary body. The third rabbit (L7) was given an injection of approximately the same amount of rabbit uveal pigment into the anterior chamber and vitreous body. Similar injections were given to control animals. All the eyes were enucleated at the end of fifteen to seventeen days.

While the reaction in the injured and inoculated eyes of the hypersensitive animals was somewhat more severe than that observed in the control animals, almost all the eyes into which injections were made still showed a mildly positive aqueous ray at the time of enucleation. The histologic picture observed in the eyes of the hypersensitive animals was essentially the same as that seen in the control animals. Large clumps of free pigment could be seen in some of the eyes (fig. 11), while others showed phagocytosis of the injected pigment by monocytes and few epithelioid cells with a rather scanty mononuclear infiltration in one of the eyes. In no part of any of the eyes could there be seen phagocytosis of the animal's own pigment, nor did any of the eyes simulate the picture commonly observed in sympathetic ophthalmia.

SUMMARY

A method of preparation of "uveal tissue toxin" is described. Repeated intracutaneous injections of bovine uveal tissue toxin produced hypersensitivity to swine, rabbit and autogenous uveal pigment in 6 of the 22 rabbits used. Similar sensitizing injections of rabbit uveal tissue toxin or of uveal tissue pigment devoid of staphylotoxin did not produce sensitization in any of the rabbits. The criterion of hypersensitivity was based on the histologic cellular reaction observed in tissues excised from the skin two weeks after the injection. Injury in the form of an iridectomy and single intraocular injections of uveal pigment in hypersensitive animals produced a histologic picture not greatly different from that observed in tissues of control animals; this reaction did not simulate the picture commonly observed in sympathetic ophthalmia.

EPITHELIAL PLAQUES OF THE CONJUNCTIVA AND THE CORNEA

JOHN V. V. NICHOLLS, M.D.
MONTREAL, CANADA

The term epithelial plaque was originated by Lister and Hancock [1] in 1903 to denote local hyperplasia and cornification of the conjunctival and the corneal epithelium. It is a rare pathologic lesion when affecting the eye. Many other names have been used in the literature for these lesions, such as tyloma (Gallenga [2]), cornification of the conjunctiva (Best [3]), keratosis (Mohr and Schein [4]) and conjunctival callosities (Saemisch [5]). However, epithelial plaque would seem to be the most desirable term, as it is noncommital with regard to the type of hyperplasia and its etiology.

It is usual for authors (Parsons [6] and Duke-Elder [7]) to include under this name hyperplasias of several types. They were first described as occurring on the cornea by Bowman [8] in 1849. Similar cases were described by Warlomont [9] in 1860 and by Hocquart [10] in 1881. Gallenga,[2]

From the Department of Ophthalmology, Royal Victoria Hospital.

1. Lister, W. T., and Hancock, W. I.: Epithelial Plaques of the Conjunctiva, Roy. London Ophth. Hosp. Rep. **15**:346, 1903.

2. Gallenga, C.: Osservazione de tiloma della congiuntiva, Gior. d. r. Acad. di med. di Torino **33**:789, 1885.

3. Best: Ueber Verhornung des Bindehautepithels (Tyloma conjunctivae), Beitr. z. Augenh., 1898, no. 34, p. 1.

4. Mohr, M., and Schein, M.: Keratosis conjunctivae, Arch. f. Augenh. **39**: 231, 1899.

5. Saemisch: Die Bindehautschwiele, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1904, vol. 5, pt. 2, p. 412.

6. Parsons, J. H.: The Pathology of the Eye, London, Hodder & Stoughton, 1904, vol. 1, pt. 1, p. 130.

7. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 1762.

8. Bowman, W.: Lectures on the Parts Concerned in the Operations on the Eye, and on the Structure of the Retina, London, Longman [and others], 1849.

9. Warlomont: Production épithélial de la conjunctive sclérotico kératique, Ann. d'ocul. **44**:253, 1860.

10. Hocquart, E.: Placques épithéliales de la cornée, Arch. d'opht. **1**:289, 1881.

Best,[3] Mohr and Schein [4] and Saemisch [5] described such lesions on the conjunctiva.

A case of epithelial plaque formation showing marked hyperplasia came to my attention at the Mandviwalla Free Eye Hospital, Karachi, India.

REPORT OF A CASE

A Mohammedan boy 14 years of age came to the clinic complaining of a growth on the right eye, which was of one year's standing and was slowly increasing in size. On examination the patient was found to be a well developed and well nourished lad of about the stated age. Ocular examination of the right eye showed a grayish white plaque astride the corneoscleral margin, directly at 9 o'clock. It was about 6 by 4 mm. in size and slightly raised, one third of it being on the cornea. The margin was well circumscribed, and there was no inflammatory reaction. The mass was only slightly movable on the sclera and the cornea. The eye was normal in every other respect.

Fig. 1.—Vertical section through the specimen, showing the greatly thickened epithelium and corium infiltrated with small round cells. Magnification, × 40; hematoxylin and eosin.

The growth was easily removed. Drops of a 4 per cent solution of cocaine hydrochloride were instilled, and the bulbar conjunctiva was incised about the growth, a collar of healthy conjunctiva about 2 mm. wide being left attached to it. The growth was then removed by blunt dissection. The base was cauterized with actual cautery. The surrounding conjunctiva was undermined and closed over the wound by three fine silk sutures. The wound rapidly healed by first intention.

The removed tissue was fixed in a 10 per cent dilute solution of formaldehyde and paraffin-embedded sections were cut. These showed a well circumscribed area of epithelial hyperplasia. The conjunctival epithelium, instead of being four or five cells thick, was twelve to fifteen cells deep. The superficial layers were keratinized. The corium was somewhat thickened. Lymphocytes and plasmocytes were scattered throughout it, being more densly packed in some areas. The subconjunctiva in the region of the limbus contains under normal circumstances scattered

round cells. In the present case these were denser than normally. The epithelial papillae were exaggerated, and in many places the epithelium had grown down in long columns into the corium. Here the epithelial cells were markedly hyperplastic, many mitotic figures being seen. However, in no place had the epithelial cells broken through the basement membrane. The picture was one of intense epithelial hyperplasia bordering on neoplasia, being essentially similar to what is called leukoplakia in other mucous membranes in the body, such as the tongue, mouth and genitalia.

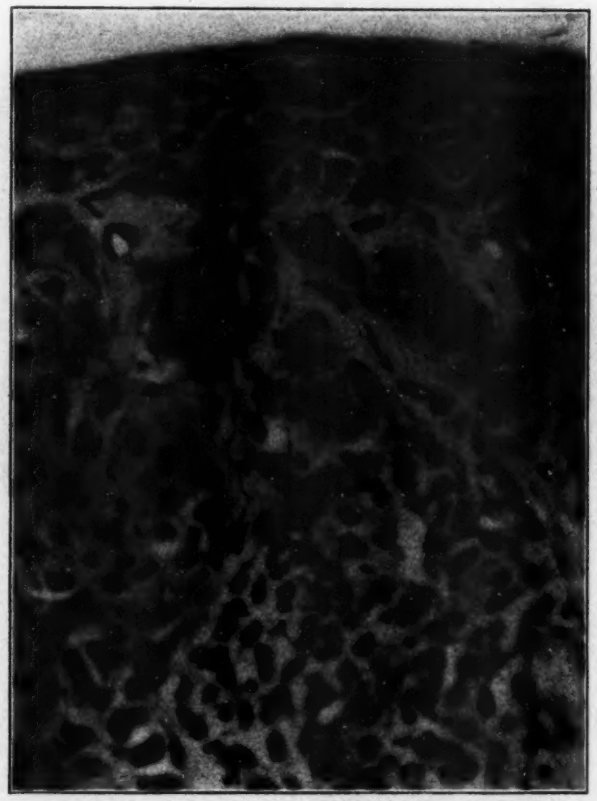

Fig. 2.—Highly magnified view of the section showing the thickened hyperplastic epithelium growing down in columns into the corium which is densely infiltrated with lymphocytes and plasmocytes. Magnification, × 400; hematoxylin and eosin.

CLASSIFICATION OF EPITHELIAL PLAQUES

Great difficulty arises in constructing an adequate classification of epithelial plaques of the conjunctiva and the cornea, because many of the entities merge. The following classification is presented with the realization that none such can be perfect:

1. The first variety occurs in patients with xerosis, no matter what may be the cause of the xerosis, whether vitamin A deficiency, con-

junctival scarring or exposure, as in ectropion and lagophthalmos. The conjunctival epithelium, either in a patchy manner or more diffusely, undergoes thickening and eventually cornification on its surface. The surface of these areas becomes rough, gray and dry. Sebaceous material becomes adherent to them. This is easily brushed off, even by the lids, and reforms in twenty-four to thirty-six hours.

2. The second, and rarer, variety is apparently congenital. It is found in young persons, having been present from birth. The lesion usually is situated astride the limbus, frequently in the position of dermoids. Parsons expressed the belief that this variety is related to the dermoids, the epidermal elements alone being represented in the tumor. He reported 2 such cases. Lister and Hancock [1] and Agricola [11] have also described cases. The lesion consists of a gray-white, slightly raised, well circumscribed plaque. It may be located at the limbus or in the typical position of dermoids, up and out. Another congenital variety has been described by Bornancini [12] and Koyanagi [13] as part of a general congenital predisposition to keratosis.

3. The third and last variety is related etiologically to some irritative condition and histologically to the leukoplakias. It is composed of a well circumscribed thickening and keratinization of the conjunctiva. The corium in some of the cases described had undergone hyaline or amyloid degeneration (Best,[3] Bihler [14] and Stock [15]). In most instances there is small round cell infiltration. The growth may occur on the corneal epithelium (Wilson [16]) or on the bulbar conjunctiva near the limbus (Heilbrun [17]). Wollenberg [18] described such a growth covered by the lower lid. In the case of Mohr and Schein [4] the lesion was under the upper lid. Usually, however, it is opposite the palpebral fissure.

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11. Agricola, B.: Ueber kongenitale epitheliale Bindehautxerose, Klin. Monatsbl. f. Augenh. (supp.) **43**:80, 1905.

12. Bornancini, V.: Cheratosi congenita dell'epitelio conjunctivale e cornale, Ann. di ottal. **39**:190, 1910.

13. Koyanagi, Y.: Beitrag zur Kenntnis der Keratosis et corneae nebst Bemerkungen über ihre Beziehung zur Xerosis epithelialis congenita, Klin. Monatsbl. f. Augenh. **69**:52, 1922.

14. Bihler, W.: Ueber epitheliale Formen der Pinguecula, Klin. Monatsbl. f. Augenh. **37**:77, 1899.

15. Stock, W.: Ueber Verhornung des Bindehautepithels (Tyloma conjunctivae), Klin. Monatsbl. f. Augenh. **66**:622, 1921.

16. Wilson, R. P.: Epithelial Placque of the Cornea, in Sixth Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, Schindler's Press, 1931, p. 52.

17. Heilbrun, K.: Ein Beitrag zur Kenntnis der Neubildungen am Limbus corneae, Arch. f. Ophth. **77**:541, 1910.

18. Wollenberg, A.: Ein neuer Fall von Bindehautschwiele, Klin. Monatsbl. f. Augenh. (supp.) **78**:135, 1927.

Plaques of this variety seem to fall into two fairly well defined groups histologically: (*a*) those in which hyperplasia is not excessive nor at all uncontrolled and (*b*) those characterized by rapid growth and marked hyperplasia. Etiologically and histologically, these subgroups seem to be two phases of the same process. It is to subgroup *b* that the specimen in the present case would seem to belong.

Best,[3] Bihler [14] and Saemisch [5] suggested that these growths are modified pingueculas; but this view is not generally accepted now. They seem to arise on the basis of some chronic irritation, as stated before. Lister and Hancock [1] reported 2 such cases, apparently following an injury from a foreign body. Elschnig [19] described such a growth occurring in association with chronic conjunctivitis. Thus, histologically and etiologically the lesions fall in the same group as the leukoplakias.

As stated before, some varieties of this group are very hyperplastic, so much so that they might be called precancerous. The lesion in Lister and Hancock's 2 cases was extremely hyperplastic, as was that in the case reported in this paper. The growth in Best's [3] case showed some marked hyperplastic features, and Wolff [20] illustrated such a growth in his book. These growths are unlike other precancerous conditions, such as the papillomas, in that they have no fibrous stalk or overhanging edges. A transitional stage has been described by Contino.[21] That epithelial plaques of this variety are precancerous is shown by the fact that they recur after roentgen treatment, as in Wollenberg's [18] case, or after scraping and cauterization, as in the case of Jacqueau and Bujadoux.[22] Yamagiwa and Ishikawa [23] produced epithelial plaques in rabbits by the local application of tar and obtained metastases. This tendency of the plaques to develop into cancer is another feature in common with the leukoplakias.

This close relation or even identity with the leukoplakias found elsewhere in the body or in various mucous membranes cannot be passed over lightly. Chichkanov [24] described a case in which he thought the condition was definitely leukoplakia. For this reason it is interesting

19. Elschnig, A.: Beitrag zur Aetiologie und Therapie der chronischen Conjunctivitis, Deutsche med. Wchnschr. **34**:1133, 1908.

20. Wolff, E.: A Pathology of the Eye, Philadelphia, P. Blakiston's Son & Co., 1934, p. 35.

21. Contino, A.: Neue Beobachtungen und Untersuchungen über die Papillome des Limbus und der Hornhaut, Arch. f. Augenh. **68**:366, 1911.

22. Jacqueau and Bujadoux: Keratosis épibulbaires à multiples récidives, Bull. et mém. Soc. franç. d'opht. **37**:329, 1924.

23. Yamagiwa, K., and Ishikawa, K.: Experimentelle Studie über die Pathogenese der Epithelialgeschwülste, Mitt. a. d. med. Fak. d. k. Univ. zu Tokyo **15**: 295, 1916.

24. Chichkanov, A. H.: Rare Case of Leucoplakia of the Conjunctiva of the Upper Lid, Rusk. oftal. zhurn. **10**:728, 1929.

to look into the pathologic characteristics of the leukoplakias. The etiologic factors leading to the production of leukoplakia and the histologic and pathologic behavior of the lesion are well known. The marked tendency for the lesion to go on to neoplasia gives one pause when one considers the treatment of epithelial plaques. Fox [25] studied 40 cases of leukoplakia buccalis; 38 of the patients were men and 2 were women. Four of the 40 patients, or 10 per cent, had a definite epithelioma, and 4 others were suspected of having it. Of 566 cases of leukoplakia buccalis recorded by Mantilla,[26] cancer eventually developed in 32 per cent. Similarily, Bloodgood [27] found that in 41 of 160 cases of cancer of the tongue there was a long-standing history of leukoplakia. The greatest predisposing factor in these cases was chronic irritation from bad teeth or from smoking a pipe. Syphilis also played a role. Of Fox's 40 patients, 65 per cent had syphilis. Though in the reported cases of epithelial plaques of the conjunctiva syphilis was so seldom looked for that no reliable figures can be arrived at, it appears that syphilis plays no significant role. Fox expressed the belief that in spite of the high incidence of syphilis in the cases of leukoplakia buccalis, it is not an essential factor. That this is the true point of view is borne out by the frequency of leukoplakia and epithelioma of the vulva arising on the basis of kraurosis and chronic irritation alone. Graves and Smith [28] reported finding leukoplakia 16 times and kraurosis 14 times in 21 specimens of vulvar epithelioma. Epithelioma of the vulva developing on a basis of leukoplakia or kraurosis has also been described by Singer,[29] Zikmund,[30] Trenz [31] and Lévy and Glasser.[32]

From the foregoing data it will be readily understood that epithelial plaques must be treated with the greatest of circumspection. An attempt should be made to classify each growth properly. The treatment will depend on the type found. Epithelial plaques of the first type rapidly disappear when the cause of the xerosis is removed. Those of the second

25. Fox, H.: Leucoplakia Buccalis, J. A. M. A. **85**:1523 (Nov. 14) 1925.

26. Mantilla, P. L.: Leucoplasies et cancer, Thesis, Paris, no. 324, 1901.

27. Bloodgood, J. C.: Cancer of the Tongue: A Preventable Disease, J. A. M. A. **77**:1381 (Oct. 29) 1921.

28. Graves, W. P., and Smith, G. V.: Kraurosis Vulvae, J. A. M. A. **92**: 1244 (April 13) 1929.

29. Singer, H.: Beiträge zur Pathologie und Therapie der Leucoplakia und des Pruritus vulvae, Zentralbl. f. Gynäk. **53**:634, 1929.

30. Zikmund, E.: Cancroid auf der Basis einer Leukoplakie und Craurosis vulvae, Zentralbl. f. Gynäk. **53**:634, 1929.

31. Trenz, M.: Epithelioma de la région clitoridienne, Bull. Soc. franç. de dermat. et syph. **35**:69, 1928.

32. Lévy, G., and Glasser, M.: Leucoplasie végétante (Kraurosis vulvae) et épithélioma de la région clitoridienne, Bull. Soc. franç. de dermat. et syph. **35**: 70, 1928.

type can be left alone or removed, depending on the surgeon's or the patient's choice in the matter. Those of the third type should always be removed as completely as possible, and any irritative condition in the conjunctival sac should be eradicated. Also, the patient should be carefully followed for any recurrence. The same is true for all cases in which the surgeon finds it difficult or impossible to classify the plaque on examination.

SUMMARY

A case of epithelial plaque of the corneoscleral margin has been described.

The literature has been reviewed in an attempt to classify these rare lesions.

The following classification has been presented: (1) epithelial plaques in patients suffering from xerosis, (2) epithelial plaques of congenital origin and (3) epithelial plaques resulting from chronic irritative conditions in the conjunctival sac. The third type may be subdivided into: (*a*) those showing cornification and epithelial hyperplasia of moderate degree and (*b*) those showing cornification and marked hyperplasia. The close similarity or even identity of the latter group with leukoplakia found elsewhere in the body is pointed out. Here the lesion seems to be a precancerous condition.

Sir Henry Holland, ophthalmologist to the Mandviwalla Free Eye Hospital, Karachi, India, gave me permission to publish this case, and Mr. C. Dee Shapland, F. R. C. S., pathologist to the Royal London Ophthalmic Hospital, had the microscopic sections prepared.

SUBCONJUNCTIVAL INJECTIONS OF NEOPRONTOSIL IN THE TREATMENT OF OCULAR INFECTIONS

R. TOWNLEY PATON, M.D.
NEW YORK

Neoprontosil, formerly called prontosil soluble or prontosil, is disodium sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate. It is employed in a 2.5 or 5 per cent aqueous solution or in tablets. Its action on the various infective organisms in the body is similar to that of sulfanilamide (paraaminobenzenesulfonamide) but not identical. Long, Bliss and Feinstone [1] assumed from results of experiments on animals that its action depends on the chemical reduction in the body. There is a relatively small amount of sulfanilamide present in the solution; on analysis neoprontosil yields only 11 grains, or 0.7 Gm., of sulfanilamide per hundred cubic centimeters of the 2.5 per cent solution.

Much has already been written on the use of sulfanilamide in the treatment of trachoma and gonococcic infections of the eye. The names of Loe,[2] Rychener,[3] Lian,[4] Kirk, McKelvie and Hussein [5] and Thygeson [6] should be mentioned for the favorable results obtained in the treatment of trachoma. The names of Newman,[7] Perry,[8] Willis,[9] Goldenbury [10] and

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From the clinic of the Manhattan Eye, Ear and Throat Hospital.

Read before the New York Academy of Medicine, Section of Ophthalmology, Feb. 20, 1939.

This work was aided by the generosity of the Medical Department of the Winthrop Chemical Co., Inc.

1. Long, P. H.; Bliss, E. A., and Feinstone, W. H.: Mode of Action, Clinical Use and Toxic Manifestations of Sulfanilamide, J. A. M. A. **112**:115 (Jan. 14) 1939.

2. Loe, F.: Sulfanilamide Treatment of Trachoma: Preliminary Report, J. A. M. A. **111**:1371 (Oct. 8) 1938.

3. Rychener, R. O.: Trachoma Treated with Sulfanilamide, Memphis M. & S. J. **13**:158, 1938.

4. Lian, S. B.: Prontosil Treatment of Trachoma, Geneesk. tidjschr. v. Nederl.-Indië **78**:1058, 1938.

5. Kirk, R.; McKelvie, A. R., and Hussein, H. A.: Sulphanilamide in the Treatment of Trachoma, Lancet **2**:994, 1938.

6. Thygeson, P.: Treatment of Conjunctivitis, Arch. Ophth. **19**:586 (April) 1938.

7. Newman, H. W.: Sulfanilamide in Gonorrheal Ophthalmia in Young Children, Texas State J. Med. **33**:585, 1937.

(Footnotes continued on next page)

L. J. and R. Fernandez [11] should be mentioned for the reports on cases of gonococcic infections of the eye in which cure was obtained with sulfanilamide or neoprontosil. Sulfanilamide has also been used with success in the treatment of inclusion conjunctivitis.[12] Loe, in treating trachoma among the Sioux Indians, reported that the dose used uniformly in this series was ⅓ grain (0.0216 Gm.) of sulfanilamide per pound of body weight per day, in divided doses, with an equal amount of sodium bicarbonate. This was continued for the first ten days; for the next fourteen days the dose was decreased to ¼ grain (0.0162 Gm.) per pound per day. Gradle, in discussing Loe's paper, is in essential agreement with him and recommends the use of sulfanilamide in the early stages of the disease. He found, as did Loe, that there was practically no improvement in persons with trachoma IV. However, all persons, regardless of the stage of the trachoma, showed a diminution of photophobia and lacrimation.

References on the use of subconjunctival injections of neoprontosil are scarce. In fact, until recently only one reference could be found. Heinz,[13] in a paper on exogenous and endogenous infections of the eye, reported a case of iridocyclitis with large exudates appearing in the anterior chamber in which cure was obtained by two subconjunctival injections of neoprontosil. The vision improved from counting of fingers to 6/6 within a week, and the anterior chamber was cleared of exudates in twenty-four hours. Brav [14] recommended topical application of a 2.5 per cent solution of neoprontosil in the conjunctival sac three times daily for the treatment of corneal ulceration. In a case of trachoma with corneal involvement improvement was noticed after five days of treatment. Glover [15] used topical applications of neoprontosil combined with oral administration of sulfanilamide for the treatment of corneal infection, with good results.

8. Perry, C. S.: Gonorrheal Conjunctivitis: Treatment with Sulfanilamide and Fever Therapy, Ohio M. J. **34**:176, 1938.

9. Willis, T.: Sulfanilamide in Ophthalmia Neonatorum, Yale J. Biol. & Med. **10**:275, 1938.

10. Goldenbury, M.: Sulfanilamide in Ophthalmology, with Report of a Case, Am. J. Ophth. **21**:54, 1938.

11. Fernandez, L. J., and Fernandez, R.: Sulfanilamide in Gonorrheal Ophthalmia, Am. J. Ophth. **21**:763, 1938.

12. Thygeson, P.: Sulfanilamide Therapy of Inclusion Conjunctivitis, Am. J. Ophth. **22**:179, 1939.

13. Heinz, K.: Subkonjunctivale Injektion von Prontosil solubile (Bayer) bei exogener und endogener Infektion des Augeninnern, Ztschr. f. Augenh. **95**:365, 1938.

14. Brav, A.: Use of Sulfanilamide in Corneal Infections, Mod. Med. **7**:84, 1939.

15. Glover, L. P.: Some Uses of Sulfanilamide in Ophthalmology, Am. J. Ophth. **22**:180, 1939.

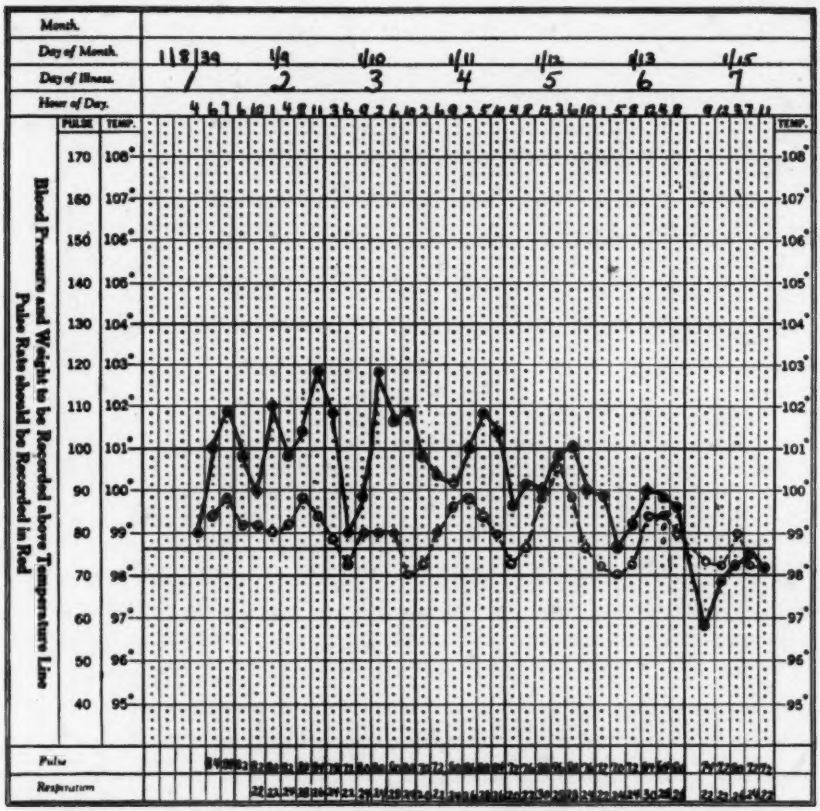
Experimental investigation on the use of sulfaniamide for ocular conditions is limited also, but the results have been no less startling. On the basis of some recent experiments on rabbits, Rambo [16] concluded in general as follows: The eye tolerates the action of sulfanilamide, and the inner structures of rabbits' eyes showed no harmful effects, even when fairly large doses were injected into the vitreous. He showed that when hemolytic streptococci are injected into the vitreous humor of a rabbit's eye; the infection could not be checked by oral administration of sulfanilamide, but when the organisms were injected into the anterior chamber, sulfanilamide administered by mouth not only checked the infection but permanently overcame it. His explanation of this difference is that the leukocytes can reach the organisms in the center of the vitreous much less readily than they can reach those in the anterior chamber. No doubt there are other factors which as yet have not been determined. Another interesting observation that Rambo made on rabbits' eyes was that the protection from streptococcic infection of the eye was as great when sulfanilamide was injected into the anterior chamber as when it was administered orally. There can be no question, from these observations and from what I have observed in the clinic, that there must be some effective mechanism brought into play with the local administration of the drug. Because of the great diffusibility of the drug, it will be difficult to determine the exact concentration necessary to combat both extraocular and intraocular infections.

TOXIC REACTIONS

In the last two years the literature on this subject has been so overwhelming that I shall do no more than give a word of warning to those who have not been accustomed to administering the drug and to caution them to be ever watchful for those persons who might show an unsuspected idiosyncrasy. There is no specific therapeutic test available to determine idiosyncrasies. Small doses should be given for a few days before larger doses are administered, and then the drug should be given only when careful hematologic follow-up work can be performed. This means hospitalization of the patient as a rule, and for slight infections this is not practical or economical. Thus far toxic reactions have not been noted when the drug has been administered subconjunctivally. An unusual idiosyncrasy to the drug, when administered orally, was shown by one of my patients. Because of a high degree of sensitivity to all forms of medication, he was given 30 grains (1.95 Gm.) of sulfanilamide by mouth daily for the treatment of superficial punctate keratitis following an infection of the upper part of the respiratory tract. On the

16. Rambo, V. C.: Effects of Sulfanilamide on Rabbits' Eyes, Am. J. Ophth. **21**:739, 1938.

fourth day the dosage was reduced to 20 grains (1.3 Gm.). On the seventh day fever and some mild gastric disturbances developed, and a slight cyanosis of the lips was noted. The accompanying chart shows the course of the temperature during the following eight days. The patient made a protracted but uneventful recovery. Laboratory tests were found to be negative, and all other possible causes for such a temperature reaction were eliminated. The sulfanilamide concentration in the blood showed only a slight increase during the febrile period. It occurred



Temperature chart of patient, showing generalized toxic reactions after peroral administration of sulfanilamide for one week.

to me that had further treatment been necessary the subconjunctival injections of neoprontosil might have been used to avoid further toxic reaction.

DOSAGE

The drug may be given either intravenously or intramuscularly. The total amount required over the first twenty-four hours of treatment is calculated on the basis of 1 cc. of a 2.5 per cent solution for each pound

of body weight up to 120 pounds (54.4 Kg.). For patients over this weight, the first day's dose is 120 cc. of a 2.5 per cent solution. The total daily amount is divided into six parts, and the fractional dose is given every four hours by subcutaneous injection. Often in the more severe infections an 0.8 per cent solution of sulfanilamide in physiologic solution of sodium chloride may be given in addition to tablets of sulfanilamide or neoprontosil. There is no available information to date on the amount of neoprontosil needed when administered subconjunctivally, but it seems from the experience at the Manhattan Eye, Ear and Throat Hospital that from 0.3 to 0.5 cc. of a 2.5 per cent solution may be given every two days for several doses without apparent injury to the tissues of the eye.

PURPOSE OF SUBCONJUNCTIVAL INJECTIONS

Subconjunctival injections of neoprontosil were first given in an effort to find a safer method of administering the drug in large clinics in which laboratory follow-up work was not possible in each and every case of ocular infection. It was also felt that in dealing with intraocular infections a more concentrated effect could be obtained at the site of the infection if this method of injection were used. This might be a deciding factor in attempting to save one eye which is severely infected, if the other eye is not involved and if large doses of sulfanilamide given by mouth might not only cause general toxic manifestations but result in poor vision in both eyes. A case of toxic amblyopia has been reported.[17] This method may also prove advantageous for patients who show an idiosyncrasy for the drug when it is given by mouth.

The peroral mode of administering sulfanilamide and the subconjunctival injection of neoprontosil appear to parallel each other as to the beneficial effect for certain ocular infections. I am not in a position as yet to say definitely that one method is superior to the other when used alone. So far in the treatment of trachoma only a few patients have received subconjunctival injections alone. The improvement in the subjective symptoms have been as follows: Lacrimation ceased within twenty-four hours of the initial injection; photophobia and blepharospasm quickly disappeared after the first or second injection. The other symptoms disappeared in a variable period of time, depending on the severity of the infections. In general, my results are similar to those reported by Loe. Credit should be given to him for the following observations: "Improvement of objective symptoms included paling of the conjunctiva and paling of the trachomatous patches and flattening of the granules and follicles. The blood vessels of the conjunctiva become more visible on the fifth and sixth day of treatment and daily thereafter

17. Bucy, P. C.: Toxic Optic Neuritis Resulting from Sulfanilamide, J. A. M. A. **109**:1007 (Sept. 25) 1937.

they become more normal." There was also an improvement in the appearance of the pannus. The granules of the lower lids were found to be the last objective symptom to disappear.

METHOD OF ADMINISTRATION

Before administering neoprontosil subconjunctivally, thorough anesthesia of the conjunctiva is obtained with a 0.5 per cent solution of pontocaine hydrochloride. An initial injection of about 0.3 cc. of neoprontosil in either the upper or the lower cul-de-sac is given by means of a tuberculin syringe. The patient will experience a sharp stinging sensation lasting about three minutes. In cases in which there is much scar tissue it is advisable to give numerous smaller injections, as the accompanying pain is not so great. In most cases the pain is evidently due to the breaking of the adhesions from the pressure of the fluid. Gentle massage of the spot where the injection is given is then performed through the closed lids, in order to hasten the dispersion and absorption of the drug. Thus far no general or local toxic reaction and no injurious effect on the structures of the eye have been observed. Usually the dye constituent of neoprontosil has been completely absorbed in two days, at which time the injection should be repeated. One of my observations which needs corroboration is that if there has been no improvement after the third injection, little if any improvement may be expected from subsequent injections.

RESULTS

Good results have been obtained in several cases of iritis, scleritis and interstitial keratitis, but owing to an insufficient number of patients treated, I do not yet feel warranted to say just what type of infection responds best to this form of chemotherapy. One patient with gonococcic iritis showed marked improvement after one injection of a 2.5 per cent solution of neoprontosil, atropine being used to dilate the pupil. Otherwise, no other treatment was given. Later the injections were supplemented by tablets, and the patient made a complete recovery.

An emaciated-looking Chinaman aged 35 staggered into the clinic late one afternoon, and because of the severity of his ocular condition was immediately admitted to the ward. It was at first thought that he was suffering from an acute exacerbation of an old trachomatous infection in both eyes. Examination revealed an edematous conjunctiva with much scarring of both upper lids. There was a generalized vascularization of the corneas, both superficial and deep; pannus, and small ulcerations, which stained readily with fluorescein. Only a few old trachomatous follicles could be seen on the tarsal surface of the lids. Vision could not be determined accurately owing to the intense photophobia and profuse lacrimation. The patient had a 4 plus Wassermann reaction and numerous badly infected teeth. The blood sugar was 150 mg. per hundred cubic centimeters. This returned to normal after the patient spent a few days in the hospital on a proper diet. On admission and during his stay in the hospital, the only form of local therapy used

were subconjunctival injections of neoprontosil and instillations of atropine sulfate. The first day he received an injection of 0.5 cc. of neoprontosil in the upper retrotarsal fold of each eye. Great improvement was noted on the following day.

Results for Some of the Forty Treated Patients

Name	Sex	Age	Diagnosis	Complication	Duration of Disease	Sulfanilamide	Neoprontosil	Combined Drugs	Result
D. D.	M	52	Trachoma III	Ulcers	10 yr. +	..	..	+	Improved
A. G.	F	55	Trachoma III	.....	5 yr.	+	..	..	Markedly improved
F. K.	F	59	Trachoma III	Ulcers	5 yr. +	..	..	+	Not improved
R. S.	F	20	Recurrent keratoconjunctivitis	Ulcers	Attacks for 4 yr.	..	..	+	Markedly improved
H. K.	M	14	Trachoma II	Vernal catarrh	3 yr.	..	..	+	Markedly improved
D. D.	M	29	Trachoma III	Ulcers	5 yr. +	..	..	+	Markedly improved
R. M.	M	30	Keratoconjunctivitis and episcleritis	Ulcers	Attacks for 2 yr.	+	..	..	Markedly improved
E. K.	M	46	Keratoconjunctivitis and uveitis	Ulcers and ectropion	8 yr.	..	..	+	Markedly improved
R. W.	F	45	Trachoma III	Ulcers	5 yr. +	..	..	+	Markedly improved
R. M.	M	47	Episcleritis	.....	1 wk.	..	+	..	Markedly improved
S. W.	M	59	Trachoma III	Ulcers	10 yr. +	+	..	..	Markedly improved
A. D.	M	32	Trachoma III	Ulcers	10 yr. +	..	..	+	Markedly improved
M. D.	F	69	Keratitis	Ulcers	1 yr.	..	+	..	Markedly improved
J. H.	M	41	Trachoma III	Interstitial keratitis (syphilitic)	Acute attack 4 days before treatment	..	+	..	Markedly improved
M. L.	M	50	Trachoma III	Ulcers and uveitis	5 yr.	+	..	..	Markedly improved
P. N.	M	39	Trachoma I	.....	1 yr.	..	..	+	Markedly improved
D. H.	F	48	Ulcer of cornea	.....	Several weeks	+	..	..	Markedly improved
F. P.	M	21	Trachoma III	Ulcers	1 yr.	+	..	..	Markedly improved
E. S.	M	30	Trachoma III	Ulcers and uveitis	5 yr. +	..	+	..	Markedly improved
W. O.	F	53	Keratoconjunctivitis	Anterior uveitis	5 yr. +	..	+	..	Markedly improved
M. F.	F	50	Chalazion	.....	1 mo.	..	+	..	Not improved
E. C.	M	45	Gonorrheal iritis	.....	Recurrent attacks for 4 yr.	..	+	..	Improved

During the next four days he received two more injections of 0.3 cc., and on the seventh day he was discharged from the hospital and was able to go about his work. Subsequent examinations in the clinic showed that I was dealing with a case of trachoma of long standing in which secondary infection from foci of infection played a dominant part. The deep vascularization was no doubt due in part to the syphilis.

It will be interesting in the future to observe the results of this form of therapy in cases of interstitial keratitis and keratitis due to focal infection. I shall perhaps then have a better understanding of this unusual result.

CONCLUSIONS

Neoprontosil when administered subconjunctivally in small doses has been well tolerated by the eye in the presence of certain severe intraocular and extraocular infections.

For ambulatory patients, especially clinical patients, for whom a daily blood count and determination of the hemoglobin and sulfanilamide concentration of the blood cannot be performed, the combined oral and subconjunctival injections lessen the danger of severe toxic effects.

NOTE.—Since this report was made, I have been using 5 per cent aqueous solution of neoprontosil with no apparent injury to eye tissues.

DISCUSSION

DR. BENJAMIN FRIEDMAN: Dr. Gilbertson and I instilled a 1 per cent solution of sulfanilamide into the conjunctival sac of one eye of persons with catarrhal conjunctivitis and used some other antiseptic in the other eye as a control. We found that the results with the sulfanilamide were at least as good, and in some cases better, than those obtained with the usual ocular antiseptics. In some cases in which conjunctivitis recurred in spite of ordinary treatment, sulfanilamide was effective, while in other cases it seemed to be ineffective. However, no harm can be done by using this drug in a 1 per cent solution, and I would recommend it in cases of conjunctivitis.

DR. S. A. AGATSTON: I should like to know what strength solution Dr. Paton used.

DR. R. TOWNLEY PATON: I used a 2.5 per cent aqueous solution, which can be obtained from some drug firms in either a 50 cc. bottle with a rubber stopper or in ampules containing 5 cc. each. Some of the patients have received as many as five injections at intervals of two days, and there has been no residual staining. The dye disperses rapidly and apparently leaves no permanent stain.

CHANGES IN ANGIOSCOTOMAS ASSOCIATED WITH INHALATION OF OXYGEN

CHARLES M. ROSENTHAL, M.D.*
BROOKLYN

In 1938 Evans and McFarland [1] showed a relation between oxygen deprivation and angioscotomas. They demonstrated a widening of the defects, the extent of which varied with the degree of oxygen deprivation. They suggested that these changes arose because of a relative anoxemia at the synapses of the first and second retinal neurons.

According to Peters and Van Slyke,[2] the hemoglobin in the arterial blood of persons free from disorders of the respiratory tract has an oxygen saturation of approximately 95 to 96 per cent at sea level. The additional amount of oxygen that can be carried by the blood is small even when the oxygen tension in the inspired air is elevated. It corresponds at most to the remaining 5 per cent of the oxygen-carrying capacity of hemoglobin plus a small amount physically dissolved in the plasma (which is proportional to the arterial oxygen tension). It is generally believed that this small increase in arterial oxygen content which can be produced by administration of oxygen is physiologically insignificant, unless the administration is much prolonged, in which case toxic effects appear.

Since the method of angioscotometry appears to be such a delicate measure of the effects of oxygen deprivation, it suggested itself as a method of determining the effects of increased oxygen tension.

Fifteen normal persons [3] (10 males and 5 females, including interns, nurses and medical students) served as subjects. The age range for this group was 22 to 35 years. The subjects were unfamiliar with the possible effects of excess oxygen on the scotomas, and thus the possibility of suggestion was avoided.

In the preliminary investigation oxygen was administered orally by means of a rubber catheter. It was soon felt that a more comfortable

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* Fellow in Ophthalmology, Long Island College of Medicine.

From the Department of Ophthalmology, Long Island College Hospital.

1. Evans, J. N., and McFarland, R. A.: Effects of Oxygen Deprivation on Visual Fields, Am. J. Ophth. 21:968 (Sept.) 1938.

2. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1932, vol. 1, chap. 12.

3. One subject (chart 1, *6*) had keratoconus; another (chart *2*, *11*) had an alternating exotropia measuring 25 prism diopters.

and convenient method was necessary. To this end a special oxygen mask [4] was used. This proved to be a more comfortable though probably not a more efficient way of administering the oxygen. The flow of oxygen was maintained at 10 liters per minute, which gave an oxygen concentration of about 100 per cent. During the latter half of the experiment final control maps were made after the withdrawal of the oxygen.

TECHNIC

Lloyd's stereocampimeter and charts were used. The method of plotting was by means of angioscotometry, as recommended by Evans [5] and regularly employed in plotting central fields at the Long Island College Hospital. The usual silver white spheres were used, ranging in size from 0.45 to 0.52 mm.

The subjects were seated before the stereocampimeter, care being exercised that they were comfortable and free from physiologic and psychologic disturbances, and the blindspot was mapped. Following the usual method, the two main superior angioscotomas were plotted, one of these encircling the fixation area and the other going superiorly and temporally (chart 1). The latter was selected because it usually shows a large physiologic scotoma,[6] and hence slight alteration in size can readily be detected.

After the subject was seated, he was instructed to indicate the disappearance of the test object by tapping. After a brief practice period he was permitted to rest ten minutes before the control map was made. During this interval the blood pressure was recorded. On completion of the control test, the oxygen mask was attached, the subject breathing an oxygen concentration of about 100 per cent for five minutes. A map was then plotted on the same chart used for the control map, the scotomas being differentiated by the use of a colored pencil. At the completion of the record, the blood pressure was measured. The oxygen mask was removed, and after five minutes, during which the subject remained quiet, a final control map was made.

RESULTS

To compare and analyze the results obtained in this experiment, it will be necessary to divide the subjects into group A, those on whom the oral catheter was used to administer oxygen; group B, those on whom the oxygen mask was used to administer oxygen, and group C, miscellaneous subjects.

For group A, 6 subjects, 5 men and 1 woman, were selected. A final control map was not made for these subjects. Nevertheless, conclusions can be drawn. In chart 1 it is shown that a definite narrowing of the plotted angioscotomas occurred for each subject after the administration of oxygen. The nasal branch showed a narrowing which

4. MSA Comfo respirator.

5. Evans, J. N.: An Introduction to Clinical Scotometry, New Haven, Conn., Yale University Press, 1938, chap. 6.

6. This physiologic scotoma may be explained as being due to a relative edema of the inferior portion of the retina as a result of its dependency.

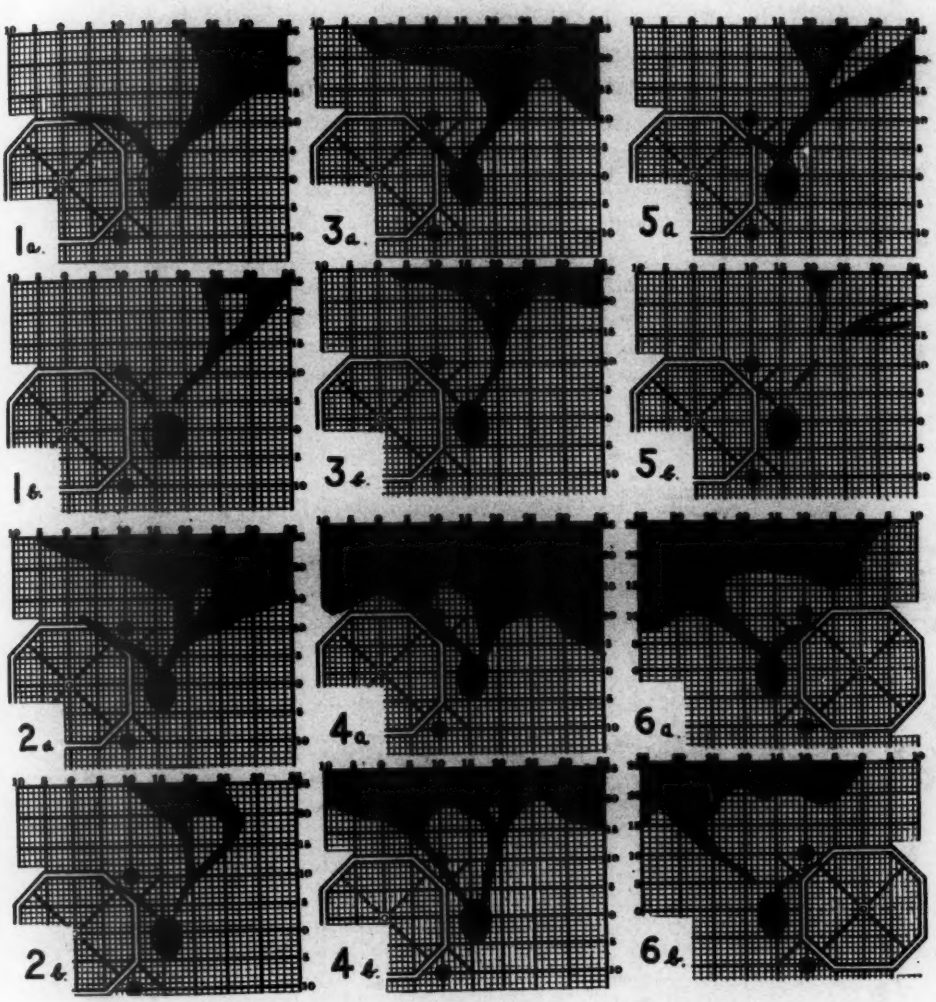

Chart 1.—Maps for the 6 subjects in group A.

Map No.	Patient	Blood Pressure: Before Inhalation	Blood Pressure: After Inhalation	Test Object	Vision in Each Eye
1	M. B.	110/75	115/80	0.45 mm.	6/6
2	M. R.	120/88	118/90	0.45 mm.	6/6
3	E. S.	115/80	120/85	0.45 mm.	6/6
4	M. W.	125/80	110/90	0.45 mm.	6/6
5	W. K.	116/72		0.60 mm.	6/6 with correction
6	M. H.	118/76		0.60 mm.	6/9 with correction

a. Before inhalation of oxygen.
b. During inhalation of oxygen (ten minutes).

varied from one-third to one-fourth the width of the original control angioscotoma. The temporal branch near its point of origin on the blindspot showed a similar change, whereas the border of the previously mentioned physiologic scotoma (superior temporal) showed a recession which varied from 3 to 12 degrees. It will be noted on several of the maps that the area which was a dense scotoma before the administration of oxygen showed islands of vision after inhalation of oxygen (chart 1, *1 b, 3 b* and *4 b*); in the other maps (chart 1, *1 b, 2 b, 4 b* and *5 b*) the area actually cleared to show dichotomous branching angioscotomas.

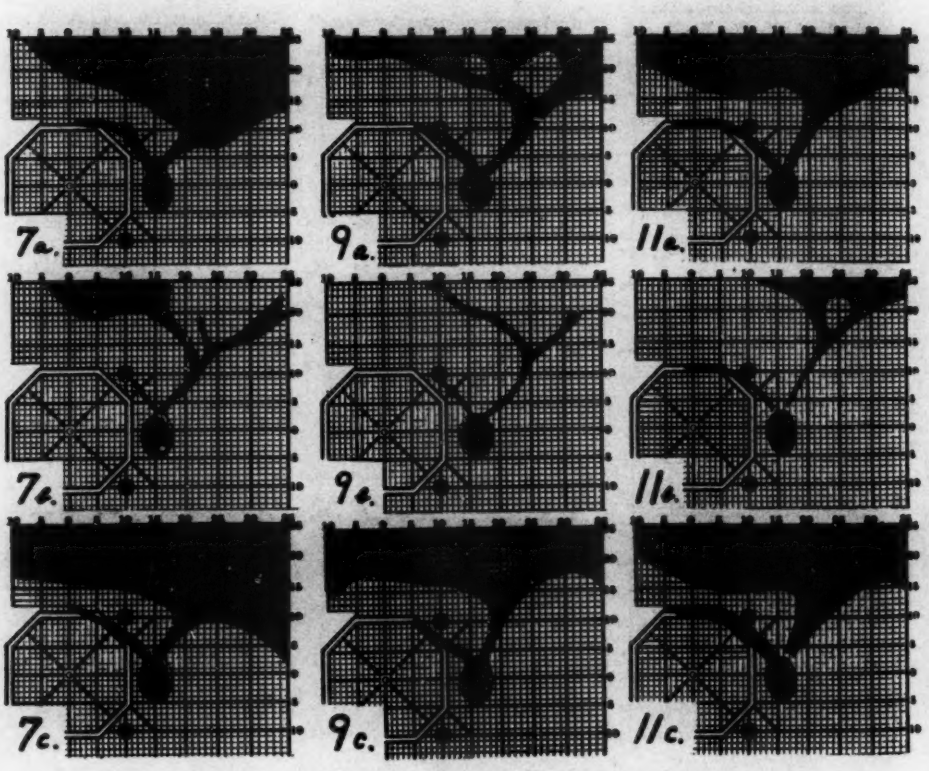

Chart 2.—Maps for 3 of the subjects in group B.

Map No.	Patient	Blood Pressure: Before Inhalation	Blood Pressure: After Inhalation	Test Object	Vision in Each Eye
7	H. A.	120/78		0.54 mm.	6/6
9	M. H.	100/70	98/70	0.40 mm.	6/6
11	R. B.	110/70	94/70	0.43 mm.	6/6 with correction

a. Before inhalation of oxygen.

b. During inhalation of oxygen (ten minutes).

c. After withdrawal of oxygen and resumption of inhalation of room air.

The dotted areas in *7c* and *9c* indicate small islands of vision which existed just prior to their becoming absolute scotomas.

It is interesting to note at this point that despite the fact that 1 subject (chart 1, *6*) was found to have keratoconus, his angioscotomas coincided with those obtained for the other subjects of this group.

The variations in blood pressure for all these subjects were negligible.

For group B, 6 subjects were also used, 4 men and 2 women. This was the best controlled group (chart *2, 7, 9* and *11;* chart 3, *8, 10* and *12*). Nevertheless, the results obtained were almost identical with those obtained with group A. The nasal angioscotomas showed a similar

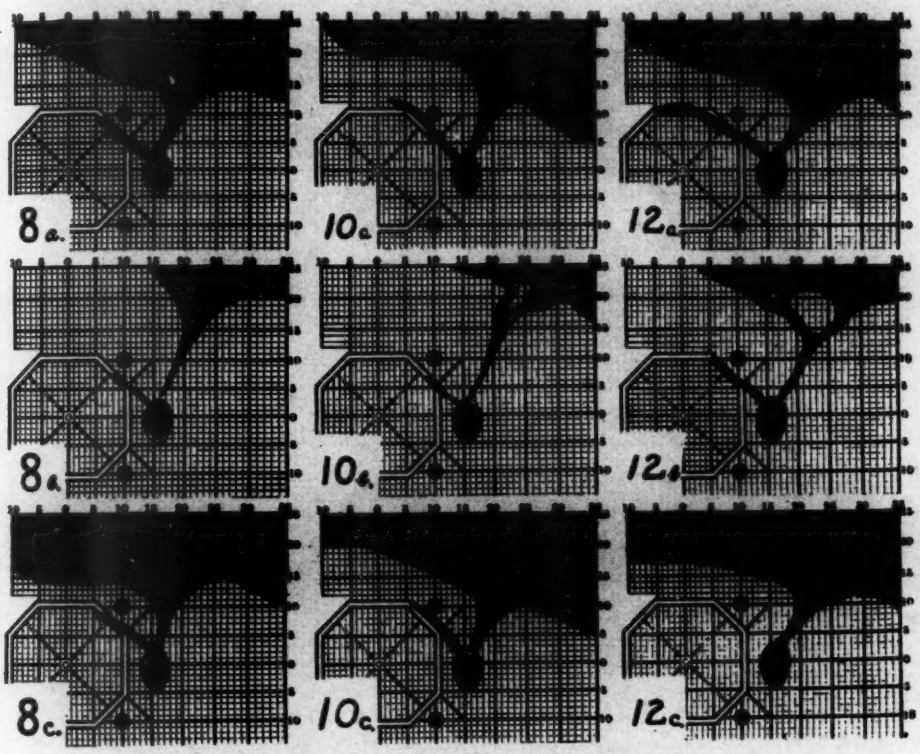

Chart 3.—Maps for 3 of the subjects in group B.

Map No.	Patient	Blood Pressure: Before Inhalation	Blood Pressure: After Inhalation	Test Object	Vision in Each Eye
8	D. M.	114/84	110/80	0.52 mm.	6/6 with correction
10	D. S.	118/74	118/74	0.52 mm.	6/6
12	S. C.	116/60	114/60	0.54 mm.	6/6

a. Before inhalation of oxygen.
b. During inhalation of oxygen (ten minutes).
c. After withdrawal of oxygen and resumption of inhalation of room air.

reduction in width of one-third to one-half their original size, whereas the border of superior temporal angioscotomas receded 5 to 10 degrees.

Similar islands of vision and dichotomous branching angioscotomas appeared (chart 2, *7, 9* and *11;* chart 3, *10* and *12*).

It appears that despite the fact that the face mask was the more desirable piece of apparatus, the results obtained by using the oral catheter were just as reliable.

After the oxygen was stopped and the subject allowed to breathe air again for four or five minutes, the final control map was plotted. Each of the subjects in this group showed a definite widening of the angioscotomas. In the superior temporal angioscotomas the widening exceeded that in the first control map by 1 degree to 5 degrees (chart 2, *7 c, 9 c* and *11 c;* chart 3, *8 c, 10 c* and *12 c*). Those few subjects

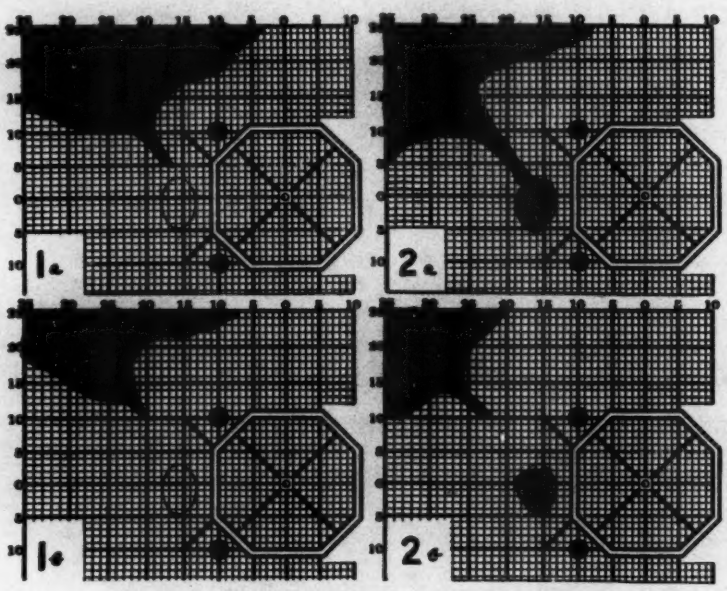

Chart 4.—Maps for the 2 subjects in group C.

Map No.	Patient	Blood Pressure: Before Inhalation	Blood Pressure: After Inhalation	Test Object	Vision in Each Eye
1	H. H.	106/84	102/80	0.45 mm.	6/6
2	M. B.	110/80	108/78	0.54 mm.	6/6

a. Before inhalation of oxygen.

b. During inhalation of oxygen.

H. H. was in the first day of her menstrual cycle. M. B. was from the clinic for patients with the toxemia of pregnancy, although at this time she was free from all symptoms of the disease.

(chart 2, *7 a* and *9 a;* chart 3, *12 a*) who had small islands of vision in the otherwise dense scotoma before inhalation of oxygen showed a disappearance of these islands in a sea of darkness after withdrawal of

oxygen. (The dotted areas in chart 2, 7 *c* and 9 *c* indicate small islands of vision which existed just prior to their becoming absolute scotomas.) One might assume that this was a secondary reaction phase.

Group C was composed of 2 subjects, both women. The first subject was in the first day of her menstrual cycle (chart 4, *1*). She exhibited the type of angioscotoma usually associated with this type of physiologic phenomenon [7] (rather extensive superior temporal scotoma). When oxygen was administered to this subject by means of the catheter, a similar narrowing of the angioscotoma occurred. The greatest amount of recession of the border of the scotoma, however, was only 5 to 6 degrees.

The other subject was selected from the toxemia clinic of the department of obstetrics (chart 4, *2*). Her map was normal in all respects. Oxygen administered by catheter again produced a response similar to that of the other patient in this group.

During the period of oxygen inhalation the areas of the defect were repeatedly checked. This process was continued after the withdrawal of the oxygen. By this means it was obvious that the narrowing was gradual, and the recovery period of widening was also gradual and continued for two or three minutes after the withdrawal of the oxygen.

CONCLUSIONS

1. Under the conditions of this experiment there was a narrowing of the angioscotomas during the period of oxygen inhalation.

2. After the withdrawal of oxygen a widening of the angioscotomas occurred which was in excess of the amount shown by the first control chart (before administration of oxygen).

COMMENT

It would seem probable from the results obtained thus far that the narrowing of the angioscotomas resulted from an increase in the oxygen content of the retina rather than elsewhere in the visual pathway. This was evident from the pattern of the narrowing, which closely followed that of the retinal vessels. Increased oxygen content of the visual pathway or cerebral cortex could not result in such changes. No definite conclusions can be drawn concerning which part of the retina was involved, although it would seem that the locus of effect was in the retinal synapse.

Since it has been shown that only an additional 5 per cent of oxygen can be added to an already oxygen-saturated blood stream (95 per cent),

7. Evans,[5] chap. 13, p. 154.

it might be justifiable to assume that the additional pressure which the oxygen was under was sufficient to cause these tissues to take up additional oxygen.

NOTE.—Since the submission of this manuscript, Bellows and Chinn [8] have reported the following observation: The oral administration of sulfanilamide resulted in a prompt accumulation of the drug in the chorioretinal layer of the eye. This portion of the eye showed the greatest concentration of the drug.

8. Bellows, J. D., and Chinn, H.: Distribution of Sulfanilamide in the Eye, J. A. M. A. **112**:20 (May 20) 1939.

RELATION BETWEEN THE VIRUS OF TRACHOMA AND THE VIRUS OF INCLUSION BLENNORRHEA

ALSON E. BRALEY, M.D.
IOWA CITY

The association of inclusion blennorrhea of the newborn with a genital disease of the mother was first discussed by Fritsch, Hofstätter and Lindner.[1] They showed that the secretion from the vagina of a mother whose baby had inclusion blennorrhea was infectious for the conjunctivas of baboons. Thygeson and Mengert [2] were able to infect the cervix of a baboon with material from a person with inclusion blennorrhea. In a previous communication [3] it was shown that the inclusion bodies found in the conjunctival epithelial cells occurred in similar types of cells in the cervix. Lindner [4] recommended the term "paratrachoma" for all diseases produced by the genital inclusion virus and suggested that they may be closely related to trachoma; he [5] expressed the belief that "paratrachoma" may represent an initial stage of mild trachoma that does not result in scarring or in pannus formation. Lindner [4] inoculated the virus of "paratrachoma" (inclusion blennorrhea) of the newborn into 21 trachomatous eyes. In most of the eyes the inoculation produced a slight added inflammation, but in those with long-standing trachoma there were no clinical signs of new infection. Superinfections of the conjunctiva of the baboon behaved in a similar manner. Thus, since one disease seems to offer protection from the other, Lindner concluded that there exists a close relation between the two types of virus, which may be similar to the relation between vaccinia and variola.

Part of a study being conducted under a grant from the John and Mary R. Markle Foundation in the Department of Ophthalmology, State University of Iowa, College of Medicine, in cooperation with the Division of Health, Office of Indian Affairs, Department of Interior, Washington, D. C.

1. Fritsch, H.; Hofstätter, A., and Lindner, K.: Experimentelle Studien zur Trachomfrage, Arch. f. Ophth. **76**:36 and 661, 1910.

2. Thygeson, P., and Mengert, W. F.: The Virus of Inclusion Conjunctivitis, Arch. Ophth. **15**:377 (March) 1936.

3. Braley, A. E.: Inclusion Blennorrhea: A Study of the Pathologic Changes in the Conjunctiva and Cervix, Am. J. Ophth. **21**:1203 (Nov.) 1938.

4. Lindner, K.: Infektionsversuche von Trachom mit Paratrachom des Neugeborenen (Einschlussblennorrhöe), Arch. f. Ophth. **133**:479, 1935.

5. Lindner, K.: Trachom und Paratrachom, Wien. klin. Wchnschr. **48**:1487 (Nov. 29) 1935.

In my experience the conjunctiva of the baboon reacts to a superinfection with either trachoma or inclusion blennorrhea with the production of acute conjunctivitis. Also, after the conjunctiva of the baboon recovers from an inclusion conjunctivitis it is susceptible to trachoma, and likewise after recovery from trachoma it is again susceptible to the virus of inclusion blennorrhea. Similarly, the conjunctiva of the baboon may again react to trachoma after having recovered from a previous infection with the trachoma virus.[6] My results confirm the findings of Julianelle, Harrison and Lange,[7] i. e., that after infection and recovery there seems to be no conjunctival immunity to either virus.

The present study was undertaken in an attempt (1) to infect the cervix of a baboon with the trachoma virus, (2) to test the virulence of cervical trachoma on the conjunctiva of the baboon during the acute infection as well as after the acute symptoms in the cervix had subsided, (3) to demonstrate inclusion bodies in the cervical epithelium, (4) to develop conjunctival immunity to the virus by cervical infection and (5) to attenuate the trachoma virus.

EXPERIMENTAL STUDIES

A patient with typical acute trachoma (MacCallan, trachom IIa) whose conjunctival material showed many free elementary bodies and inclusion bodies in the conjunctival epithelial cells was chosen for source of material. Material from this source has been used previously[8] and was known to produce experimental trachoma in a baboon.

The cervix[9] of each of 4 baboons was exposed with a glass speculum and inoculated directly with conjunctival scrapings from the patient with trachoma.

One baboon died of bronchopneumonia three days after inoculation, and at necropsy the vagina was found to be filled with a creamy white discharge, but there was no evidence of inflammation of the cervix. Smears of the discharge contained many desquamated epithelial cells but no bacteria. Epithelial scrapings of the cervix and transitional zone showed many initial body inclusions but only one elementary body epithelial inclusion (fig. 1). Histologically, the cervical epithelium was hyperplastic and the cells were swollen. The hyperplasia of the epithelium must have been tremendous, because of the large amount of epithelium on the surface of the cervix and in the discharge; however, little or no leukocytic response was in evidence in the subepithelial tissues.

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6. Braley, A. E.: Unpublished data.

7. Julianelle, L. A.; Harrison, R. W., and Lange, A. C.: Studies on Inclusion Blennorrhea: II. Experimental Transmission, Am. J. Ophth. **21**:1137 (Oct.) 1938.

8. Braley, A. E.: The Rickettsia Question in Trachoma: II. The Louse as a Possible Disseminating Agent for the Virus, Arch. Ophth. **22**:262 (Aug.) 1939.

9. The cervix of a baboon is similar histologically to the human cervix, and it was necessary to place the material at the transitional epithelial zone just inside the external os.[2]

The 3 remaining baboons were examined on the fifth day. Each showed a marked vaginal discharge and redness of the cervix. Epithelial scrapings of the cervices showed a moderate leukocytic response but no inclusion bodies in the epithelial cells. (It is not always possible to obtain satisfactory scrapings from the cervix of the baboon.)

The cervices were examined at intervals, and signs of inflammation increased in severity until about the twentieth day, when they became stationary. At no time were inclusion bodies present in the epithelial smears.

On the eighth day scrapings were taken from the cervix of one of the baboons and inoculated directly on the conjunctiva of the same animal. Five days later a mild conjunctivitis with some discharge developed. On the fifteenth day after inoculation there were slight ptosis of the upper lid, moderate purulent discharge and marked redness of the conjunctiva. Although follicles were present in the upper fornix and retrotarsal fold, smears of conjunctival epithelial cells failed to reveal inclusion bodies. Eight months after inoculation of the animal the conjunctiva continued to show the typical appearance of experimental trachoma (fig. 2).

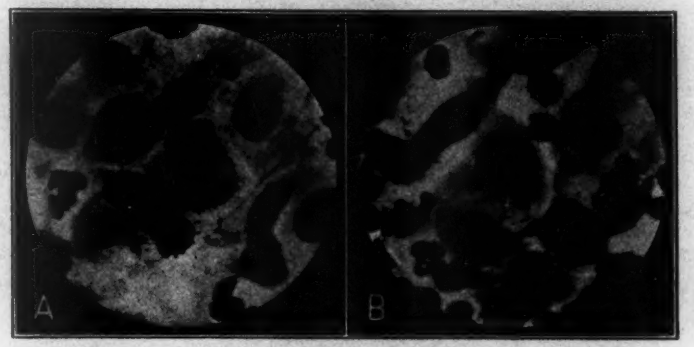

Fig. 1.—*A*, elementary body inclusion in a cervical epithelial cell. *B*, initial body inclusion in a cervical epithelial cell.

Cervical scrapings from the second baboon were transferred to the conjunctiva twenty-nine days after inoculation of the cervix. Five days later there was mild hyperemia of the conjunctiva, and in ten days follicles were present in the retrotarsal fold. Experimental trachoma developed (fig. 3 *A*), with a moderate discharge and many conjunctival follicles. The course of the infection was more acute than in the first animal, and the conjunctiva was completely healed after six months.

The conjunctiva of the third baboon was also inoculated with cervical material on the twenty-ninth day after transfer of trachomatous material to the cervix. Follicular conjunctivitis (fig. 3 *B*) and slight ptosis developed. Healing was complete in five months. The animal died three months later, and at necropsy there were still some signs of cervicitis at the transitional zone of epithelium, although there were no evidences of conjunctivitis. No inclusion bodies could be demonstrated in scrapings.

The 2 living animals were examined by a gynecologist nine months after inoculation. He reported an erosion in each baboon at the external os of the cervix similar to the lesion in human beings known to gynecologists as "congenital erosion of the cervix." No inclusion bodies were found in smears from the cervix of these baboons.

COMMENT

The primary hyperplasia of the cervical epithelium during the incubation period of the induced infection is similar to the hyperplasia produced by several proved epitheliotropic viruses. Rivers [10] and others

Fig. 2.—Experimental trachoma in baboon 2.

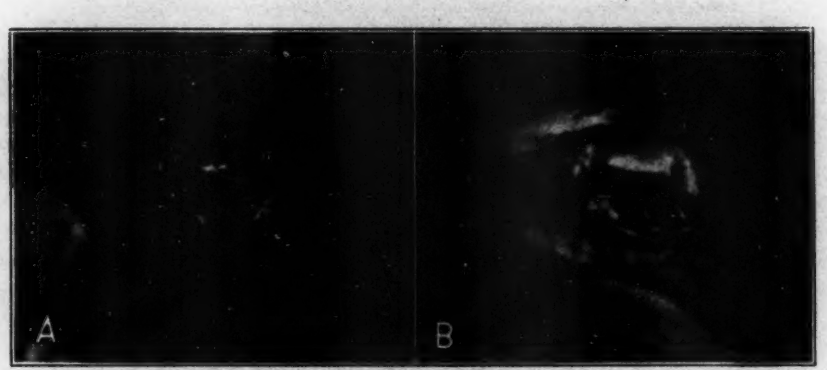

Fig. 3.—*A*, experimental trachoma in baboon 3. *B*, experimental trachoma in baboon 4.

have shown that many cutaneous and epithelial viruses produce an initial cellular hyperplasia. Hyperplasia of the conjunctival epithelium has also

10. Rivers, T. M., cited by Topley, W. W. C., and Wilson, G. S.: The Principles of Bacteriology and Immunity, ed. 2, Baltimore, William Wood & Company, 1937.

been noted in cases of inclusion blennorrhea on the first day of symptoms. Pulvertaft [11] and Julianelle [12] found that the initial response to trachomatous infection of the conjunctiva is a proliferation of epithelium.

Since the epithelial inclusions found in the cervix are predominantly initial body forms, it seems probable that a morphologic change occurs in the virus, as the cervix was inoculated with material containing chiefly elementary bodies. The initial bodies may represent a stage in the life cycle of the virus, as suggested by Thygeson,[13] or they may represent an actual increase in the size of the elementary body when it is infecting a new cell. A similar phenomenon is known to occur in bacteria during the first few hours of growth.[14]

The relation between vaccine virus and the virus of smallpox has been discussed by many writers since Jenner's time. The smallpox virus when passed through several calves becomes the vaccine virus, but it has been difficult to reverse this procedure.[15] It was thought that after inoculation of the cervix with the trachoma virus a disease similar to inclusion blennorrhea could be produced by inoculation from the cervix to the conjunctiva. However, a disease more like experimental trachoma developed in all instances. Even after twenty-nine days in the cervix the virus still produced experimental trachoma in the baboon.

Both the virus of trachoma and that of inclusion blennorrhea produce follicular conjunctivitis in monkeys and baboons (Lindner,[16] Thygeson,[17] Julianelle and his co-workers [7] and others). These authors stated that the two diseases are indistinguishable in the baboon, but, as was pointed out by Julianelle and his co-workers [7] and by Thygeson,[13] experimental trachoma is of longer duration than experimental inclusion blennorrhea. In my experience the conjunctivas of most baboons with experimental inclusion blennorrhea are healed within two months, while experimental trachoma persists for from six months to a year.

Failure to demonstrate inclusion bodies in epithelial scrapings does not definitely indicate their absence in the cervix, since only a narrow

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11. Pulvertaft, R. J. V.: The Nature of the So-Called Follicles in the Second Stage of Trachoma (Trachoma IIa), Rev. internat. du trachome **13**:19 (Jan.) 1936.

12. Julianelle, L. A.: The Etiology of Trachoma, New York, Commonwealth Fund, Division of Publications, 1938.

13. Thygeson, P.: The Nature of the Elementary and Initial Bodies of Trachoma, Arch. Ophth. **12**:307 (Sept.) 1934.

14. Topley, W. W. C., and Wilson, G. S.: The Principles of Bacteriology and Immunity, ed. 2, Baltimore, William Wood & Company, 1937.

15. Rivers, T. M.: "Filtrable Viruses," Baltimore, Williams & Wilkins Company, 1928.

16. Lindner, K.: Gonoblenorrhöe, Einschlussblennorrhöe und Trachom, Arch. f. Ophth. **78**:345, 1911.

17. Thygeson, P.: The Etiology of Inclusion Blennorrhea, Am. J. Ophth. **17**: 1019 (Nov.) 1934.

zone of epithelium is involved in the inflammation and the inclusion bodies are usually found in the deeper layers of epithelial cells. Thygeson [13] and Julianelle [12] agreed that it is also difficult to demonstrate inclusion bodies in the conjunctivas of baboons after the inflammation is manifest.

It is impossible at this time to postulate the relation between the virus of trachoma and that of inclusion blennorrhea. Perhaps the two viruses had a common ancestry and are now widely separated. If the genital inclusion virus represents an altered, avirulent trachoma virus, then inclusion blennorrhea would appear to be common among the American Indians; yet inclusion blennorrhea is almost unheard of in Indian infants. It seems that the best approach to the problem is through the human genital disease.

SUMMARY

The virus of trachoma, like the virus of inclusion blennorrhea, produces a cervicitis in baboons which when transferred to the conjunctiva produces follicular conjunctivitis. Both viruses have an affinity for the same types of epithelium and produce an initial epithelial hyperplasia. This remarkable affinity for certain cells is also exhibited by other known viruses, such as the virus of rabies and of poliomyelitis. No conjunctival immunity is produced by infection in the cervix in either disease. The virus of inclusion blennorrhea produces a short infection, while the virus of trachoma gives rise to a prolonged infection. There does not appear to be any difference in the severity of symptoms produced by the two viruses. Since the two viruses produce a similar pathologic condition, a similarity in their nature appears probable.

CONCLUSIONS

1. The virus of trachoma can produce a cervicitis in the baboon, as does the virus of inclusion blennorrhea, and this may indicate a close relation between the two viruses.

2. The immediate response of the virus appears to be the production of large forms, i. e., the initial bodies.

3. The epithelium becomes hyperplastic and hypertrophic before any evidence of inflammation is manifest.

4. The virus of trachoma does not appear to be attenuated after growth in the cervix.

5. There is no conjunctival immunity (insusceptibility) conferred by infection of the cervix with the virus.

6. The conjunctival epithelium and the transitional epithelium of the cervix appear to be the only tissues susceptible to infection with the virus of trachoma.

FORM AND POWER OF CONTACT LENSES

JOSEPH I. PASCAL, M.D.
NEW YORK

Although this paper concerns the form and power of contact lenses, what I have to say holds true of regular lenses as well. The reason, or rather reasons, why I specify contact lenses are these: In the first place, while the oddities in lenses which I am going to mention are theoretically true of all lenses, practically they are feasible mainly in the small, sharply curved contact lenses. In the second place, it was while talking about contact lenses with a number of physicians that I learned of a few erroneous ideas commonly held regarding lenses in general and contact lenses in particular. It was to point out and correct these errors that I was prompted to present this subject. Incidentally, there is a good reason why physicians who have not gone into the theory of lenses extensively should get a few false notions about contact lenses, and this is that the contact test lens is so different from the ordinary test lens. The matter of size, in that the ordinary test lens is about 37 mm. in diameter and the contact test lens is only about 12 mm. (I refer of course only to the corneal portion), is of little optical significance. But there are two differences which are significant.

1. The ordinary test lens is either double curved, i. e., double convex or double concave, or plano curved. The contact test lens is always a deep meniscus. 2. The ordinary test lens has definite power, plus or minus. The contact test lens in the ordinary set has no power; i. e., it is plano in effect. I have heard it said that the contact lens has no power because its surfaces are parallel. This is not true. If its surfaces were parallel it would have concave power; i. e., it would be a minus lens. I have also heard it said that the contact lens has no power because its two surfaces, the convex and the concave, have equal radiuses. This likewise is incorrect. If the two surfaces had equal radiuses the lens would have convex power; i. e., it would be a plus lens. The contact test lens is neither concave nor convex in effect.

To avoid any misunderstanding, I shall define my terms. A lens is plano in form if its thickness at the center is the same as its thickness at the edges. A lens is convex in form if its thickness at the center is more than its thickness at the edges, and a lens is concave in form if its thickness at the center is less than its thickness at the edges. The

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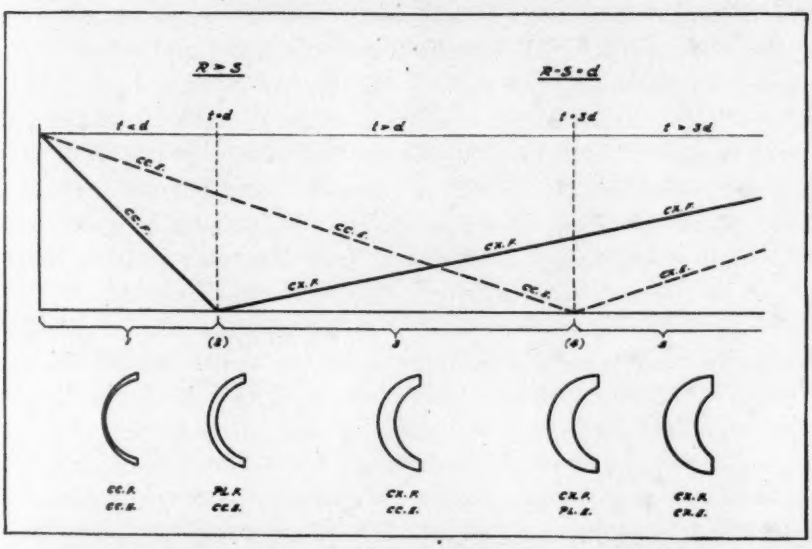
thickness in all cases is assumed to be measured in the simplest, most natural way. So much for the form of a lens. Now as to the power or effect. A lens is plano in effect if it neither converges nor diverges parallel light or if it shows no apparent motion of objects on being moved before the eye. A lens is convex in effect if it converges light or shows an "against" motion. A lens is concave in effect if it diverges light or shows a "with" motion. These definitions hold true for lenses of all shapes, but I shall confine my presentation to meniscus lenses only, this being the shape of all contact lenses.

The two surfaces of every spherical lens are sections or parts of spheres. The meniscus form of lens is produced by the intersection of two spheres. By using disks to represent these spheres, I am able to study the lenses resulting from their intersection.[1] If I intersect two spheres of unequal radiuses, I create a meniscus lens in which the outer convex radius, which I shall call R, is less than the inner concave radius, which I shall call S. I get a lens which is convex in form and convex in effect (lens A). This is true in all cases in which R is less than S, whether the lens is thin or thick. If I similarly intersect two spheres of equal radiuses I get a lens which is likewise always convex in form and convex in effect (lens B). The contact test lens having no power is evidently not made with equal radiuses. There are three possibilities as to the relative lengths of the two radiuses R and S; i. e., R is either less than S, is equal to S or is more than S. Lens A has R less than S. This is the usual way of making a convex meniscus. Lens B has R equal to S. Both of these lenses are always convex in form and convex in effect. But with the third possibility, i. e., when R is more than S, a variety of conditions is obtained.

I shall designate the difference between the two radiuses, the numerical difference between R and S, as d, i. e., $R - S = d$, and I shall designate the thickness of the lens by t. If I intersect two spheres of unequal radiuses, so that in the resulting lens the outer convex surface has a longer radius than the inner concave surface, that is, $R > S$, I get, if I make the lens extremely thin, a lens like lens 1 in the accompanying graph. In this lens t is very small, i. e., $t < d$; e. g., if the convex radius R is 9 mm. and the concave radius S is, say, 8 mm., d, the difference between the radiuses, is 1 mm. and t is less than 1 mm. Here I get a lens which is concave in form and concave in effect. This is the usual way of making a concave meniscus lens. If I increase the thickness by sliding the spheres until the thickness equals the difference between the radiuses, the two spheres become concentric. Here $t = d$. The resulting lens has parallel surfaces and is plano in form but is

1. Models of intersecting spheres and the resulting lens shapes were used in the presentation.

concave in effect (lens 2 in the graph). A contact test lens of no power evidently cannot be made in this way. If I increase the thickness still more, but not too much, by sliding the spheres still more, I get a lens which is convex in form but still concave in effect (lens 3 in the graph). Here I have a meniscus lens which is convex in form and concave in effect. If I increase the thickness still more until the thickness is three times the difference between R and S, i. e., $t = 3d$ (approximately), I get a lens which is convex in form and plano in effect (lens 4 in the graph). This is the way the contact test lens is made, convex in form and plano in effect. If the inner concave radius is, say, 8 mm. and the lens is 0.50 mm. thick, the outer convex radius



Graphic illustration of the relative changes in the form and effect of a meniscus lens of unequal radiuses, $R - S = d$, as the thickness increases from $t = 0$ to $t > 3d$.

is longer by about a third of the thickness, i. e., by 0.17 mm. If I increase the thickness of the lens still more by sliding the spheres so that $t > 3d$, I get a lens which is convex in form and convex in effect (lens 5 in the graph). Here I obtain the oddity of a meniscus lens in which the inner concave radius is shorter than the outer convex radius, which makes the inner concave surface stronger than the outer convex surface, and yet the lens is convex both in form and in effect.

By such a demonstration the forms of these lenses, these oddities in lenses, that is, whether they are plano, concave or convex, is evident. It can also be seen how they change in form from concave to plano to convex. In fact, all these forms can be obtained and their development demonstrated, so to say, by sliding three coins about in the manner I described, using, say, two pennies and a quarter. But the effect

of these lenses, that is, how they change from concave to plano to convex in effect, can be demonstrated either by trying out lenses so made or by calculation. The calculations are fairly easy, but space will not permit me to go into them now, so for the present my word for these changes in effect will have to be accepted.

I have summarized these oddities in lenses in the accompanying graph just to present a complete visual picture. Here R and S equal two fixed radiuses, with R more than S and d the difference between them. The horizontal (abscissa) line represents increasing thickness, t, starting on the left with t = zero, increasing from t less than d, to t = d, to t more than d, to t = three times d (approximately), to t more than three times d.

Starting with t = zero (as a limiting value), one gets a lens which has the highest potential concavity in both form and effect. As t increases, but still being less than d, the concavity diminishes. Through it all one has a lens (lens 1) which is concave in form (solid line) and concave in effect (dashed line). As the thickness increases until t = d, lens 2 is obtained, a lens which is plano in form but still concave in effect. As the thickness increases still more, so that t is more than d (but less than 3d), lens 3 is obtained, which is convex in form but still concave in effect. As t increases still more, until t = 3d (approximately), lens 4, which is convex in form but plano in effect, is obtained. This, by the way, is the form of the contact test lens. As the thickness increases still more, until t is more than 3d, lens 5 is obtained, a lens which is convex in form and convex in effect. It is thus shown in the graph how, by merely changing the thickness, a meniscus lens with fixed radiuses changes from a lens which is concave both in form and in effect (lens 1) to a lens which is finally completely the reverse, being convex both in form and in effect (lens 5). In between are a lens which is plano in form and concave in effect (lens 2) and a lens which is, oddly enough, convex in form and concave in effect (lens 3) and a lens which is convex in form and plano in effect (lens 4). The graph shows that the concave effect persists much longer than the concave form. The latter is lost as soon as t = d, but the concave effect persists until t = 3d (approximately). The t = 3d is derived from the relation $t = \frac{n}{n-1} \times d$; if one takes n = 1.5, then $t = \frac{1.5}{0.5} \times d = 3d$.

Another graph can be drawn to show these changes from the other end, so to say. If the thickness t is kept fixed but d changed, by starting with a lens in which d = o, i. e., R = S, a lens is obtained which is convex in form and convex in effect. By increasing d, e. g., by keeping S fixed but increasing R, less and less convexity is obtained, in both form and effect, until finally a lens is obtained which is concave both in form and in effect. By starting from this end it will be found that the convex form persists longer than the convex effect.

SUGGESTIONS FOR A NEW DESIGN OF STOCK CONTACT LENSES

HARRY EGGERS, M.D.
NEW YORK

It may fairly be stated that the stock ground contact lenses with spherical scleral shapes cannot be worn successfully by over 90 per cent of all persons. This means comfortable wear, without irritation, for a period of four hours or longer.

One reason for this failure is the fact that the contour of the anterior sclera usually is paraboloid and not spherical. The spherical scleral band of the contact lens almost never conforms to the shape of the eye. Only a very narrow, almost linear band of contact, usually the edge of the lens, exists between the sclera and the lens. The entire pressure from the lids is transmitted along this narrow rim of contact and results in an impression of the sclera and discomfort. The whole width of the scleral portion of a lens need not be in apposition to the eye, but the band of contact should be wide enough to prevent impression of the eye. There must not be any area or point of excessive pressure.

Another prominent cause of discomfort is contact with the cornea. No portion of the lens may touch the cornea or the limbus.

In some instances, even though a lens makes a broad scleral contact without undue pressure and does not touch the cornea, it still cannot be worn for reasons not yet known. Very likely the solutions now in use are unsuitable, because they were designed with only the thought in mind of imitating the osmotic pressure, and in some instances also the alkalinity, of the lacrimal secretion. It is known that the cornea obtains some of its nutrition from the tears. Present solutions do not meet any nutritional needs of the cornea. Occasionally one sees a person who is able to wear a loosely fitting movable contact lens for many hours at a time. Possibly this can be explained by the fact that such a lens quickly allows its solution to mix with, and become replaced by, the normal lacrimal secretion.

In my experience contact lenses made from casts can be worn comfortably by about 40 per cent of those who have them made. Some of the failures are caused by inaccurate fits, so that either the cornea is touched or the sclera is impressed. Other failures again are caused by unknown factors. The great objection to the molded lenses is that a patient first must go to the considerable expense and trouble of obtaining them before he can find out whether he can wear them.

A more suitable type of contact lens would be a stock type of much greater applicability than the present spherical ones. Stock lenses can be tried in advance to determine how long they can be tolerated. If a patient can wear a trial lens continuously for five hours or longer without irritation, the final lens will duplicate this performance provided an identical scleral curve and a fairly similar corneal curve have been ordered.

Examination of about 200 casts, some of them my own and the others lent to me by Mr. Theodore E. Obrig, quickly revealed one reason for the failure of stock spherical lenses. Almost all of the casts have a parabolic profile and definitely not a spherical one. Consequently, the contours of the lens and the eye do not agree. Usually only the edge of the spherical lens rests against the sclera and is pressed into it by the eyelids. If stock lenses with paraboloid shapes were available, there would be a broader bandlike area of contact between the lens and the eye and consequently much less pressure at any one place.

Another significant finding in the casts is that the profile of a cast seldom is symmetric. The profile of the nasal side in particular is different from the profiles of the other three sides—temporal, superior and inferior. This variation can be described best in a mathematical manner. The general equation of a parabola is: $y^2 = 2px$. This equation, with the corresponding proper value for the quantity 2p, will describe fairly well the curvature of the profile of each of the four surfaces of a cast of an eye. In the curve for the nasal profile the quantity 2p almost always will be appreciably larger than in the curve for the corresponding temporal profile. Also, in about 80 per cent of the casts 2p is slightly smaller numerically in the curves for the superior and the inferior profile than it is in the curve for the corresponding temporal profile. The upper and lower surfaces are much more like the temporal in contour than the nasal. In about one third of the casts the upper and lower surfaces have practically identical outlines. In about 60 per cent of the remaining two thirds of the casts, 2p is slightly greater in the curve for the profile of the upper surface.

If a number of standard permanent steel models were constructed embodying these observations, such models could be used for the manufacture of new stock lenses. The same means by which molded lenses now are obtained from plaster models could be used for obtaining the new lenses from the steel models.

Both the size and the shape of a parabola are variable and depend on the scale to which a particular curve has been drawn. Whenever it is stated that a certain contour can be described by a particular parabolic equation, it is necessary to mention the scale to which the parabola has been drawn. I have found that the contours of most of the casts can be approximated by two series of curves, one drawn to the scale of 1 unit of length equaling 2 mm. and the other drawn to the scale of 1 unit

of length equaling 2.5 mm. These curves are the parabolas represented by the following equations: $y^2 = 7x$; $y^2 = 7.3x$; $y^2 = 7.5x$; $y^2 = 7.8x$; $y^2 = 8x$; $y^2 = 8.3x$; $y^2 = 8.6x$; $y^2 = 9x$; and $y^2 = 9.5x$.

On the basis of the foregoing observations, I would suggest that the series of stock lenses presented in the accompanying table be made available in two sizes—2 mm. equal 1 unit and 2.5 mm. equal 1 unit. The curves of the four profiles are given, the numbers referring to the quantity 2p in the equation $y^2 = 2px$. The temporal profile will be the most suitable for the master profile.

These figures represent a minimum of 70 trial lenses. It is suggested further that the lenses be made 22 mm. long in the horizontal diameter and 21 mm. in the vertical. The corneal segment need have a

Sizes of Suggested Standard Contact Lenses

Lens Number	Temporal Profile	Nasal Profile	Superior Profile	Inferior Profile	Lens Number	Temporal Profile	Nasal Profile	Superior Profile	Inferior Profile
1	7.0	7.8	7.0	7.0	19	7.8	8.6	7.5	7.8
2	7.3	7.8	7.3	7.3	20	7.8	9.0	7.8	7.8
3	7.3	7.8	7.3	7.0	21	8.0	8.3	8.0	8.0
4	7.3	7.8	7.0	7.3	22	8.0	8.6	8.0	8.0
5	7.3	8.3	7.3	7.3	23	8.0	8.6	7.8	7.8
6	7.3	8.3	7.3	7.0	24	8.0	9.0	8.0	8.0
7	7.3	8.3	7.0	7.3	25	8.0	9.0	7.8	7.8
8	7.5	8.0	7.5	7.5	26	8.0	9.0	8.0	7.8
9	7.5	8.0	7.3	7.3	27	8.0	9.0	7.8	8.0
10	7.5	8.0	7.5	7.3	28	8.0	9.5	7.8	7.8
11	7.5	8.0	7.3	7.5	29	8.3	8.6	8.3	8.3
12	7.5	8.6	7.5	7.5	30	8.3	8.6	8.0	8.0
13	7.5	8.6	7.5	7.3	31	8.3	9.0	8.0	8.0
14	7.5	8.6	7.3	7.5	32	8.3	9.5	8.0	8.0
15	7.5	9.0	7.3	7.3	33	8.6	9.0	8.3	8.3
16	7.8	8.3	7.8	7.8	34	8.6	9.0	8.6	8.6
17	7.8	8.3	7.5	7.5	35	8.6	9.5	8.3	8.3
18	7.8	8.6	7.8	7.5					

diameter of only 8 or 9 mm. This is adequate for optical purposes, since the pupil is almost never as large. A larger diameter would make the corneal segment protrude too much and thereby increase its visibility. The corneal profile of the casts usually does not extend as far laterally at the limbus as the contiguous scleral profile. Therefore, any paraboloid that fits over the sclera will not be likely to touch the cornea. The trial lenses should have corneal radiuses of curvature varying from 6.6 to 10.0 mm., but with most of the curves in the range from 7.0 to 9.6 mm. The scleral and corneal identifying numbers should be stamped on the nasal end of the horizontal meridian of the scleral portion of the lens. They can then be used for orientation in inserting the lenses.

The preceding observations and suggestions are published in the hope that some manufacturer will use them as a basis for a much needed new design of standard contact lenses.

DETERMINATION OF SULFANILAMIDE IN AQUEOUS AND VITREOUS AFTER CONJUNCTIVAL AND ORAL ADMINISTRATION

WILLARD G. MENGEL, M.D.
PHILADELPHIA

Sulfanilamide (paraaminobenzenesulfonamide) has been used extensively for systemic infections of various types and has been especially valuable when streptococci, pneumococci and gonococci were found. Prior to 1936 there was no mention of sulfanilamide in the ophthalmic literature, but recently numerous papers have appeared relating to this drug. Reports on the value of sulfanilamide in ocular infections have been made by L. J. and R. F. Fernandez,[1] Rambo,[2] Mullen and others. Engelfried [3] reported that after the oral administration of sulfanilamide to rabbits the drug was found to be widely diffused throughout the body. Rambo found sulfanilamide in the aqueous, lens and vitreous of rabbits' eyes; he reported the beneficial effect of the drug when cultures of hemolytic streptococci were injected into the vitreous and aqueous of rabbits' eyes. However, a search of the available literature revealed no reports on the determination of sulfanilamide in the aqueous and vitreous of human eyes after its administration by mouth or by instillation into the conjunctival cul-de-sac.

This report does not deal with the therapeutic effect of sulfanilamide but concerns only its concentration in the aqueous and vitreous after administration in the usual doses by mouth. Sulfanilamide has been used therapeutically by local instillation into the conjunctival cul-de-sac without definite proof of its absorption by the ocular structures. This paper also deals with the question of absorption of sulfanilamide when applied locally to the conjunctiva.

Read before the College of Physicians, Philadelphia, Section on Ophthalmology, March 16, 1939.

This report is from the service of Dr. J. Milton Griscom, attending surgeon to the Wills Hospital.

1. Fernandez, L. J., and Fernandez, R. F.: Sulfanilamide in Gonorrheal Ophthalmia: A Preliminary Report, Am. J. Ophth. **21**:763 (July) 1938.

2. Rambo, V. C.: Effects of Sulfanilamide on Rabbits' Eyes, Am. J. Ophth. **21**:739 (July) 1938.

3. Engelfried, J. J.: Observations on the Absorption, Distribution and Excretion of Sulfanilamide in Normal Rabbits: A Preliminary Report, Univ. Hosp. Bull., Ann Arbor **4**:4 (Jan.) 1938.

Sulfanilamide is a relatively insoluble drug in aqueous solution. A saturated solution is less than 0.8 per cent. On the other hand, it is one of the most diffusible drugs known. The effectiveness of sulfanilamide is obtained by maintaining a definite concentration of the drug in the fluids and tissues of the body. According to Lockwood and his co-workers,[4] sulfanilamide has no effect on the formation of immune bodies. They concluded that the capacity of the micro-organism to invade tissue is strikingly depressed by sulfanilamide.

EXPERIMENTAL INVESTIGATIONS

The investigations for the determinations of sulfanilamide were made on blind eyes. One eye had absolute glaucoma with high tension, requiring repeated paracentesis or sclerotomy; the other eyes had phthisis bulbi, requiring enucleation. In one of these the vitreous was withdrawn immediately before enucleation and in the other immediately after removal of the eye. The aqueous and liquid vitreous in all eyes were obtained by withdrawing the fluid from the eye with an ordinary syringe and needle. The local anesthetic used was pontocaine hydrochloride. The determinations[5] were made for free sulfanilamide according to the method of Marshall[6] and of Marshall and Litchfield.[7]

Determination of Sulfanilamide in the Aqueous After Instillation in the Cul-De-Sac.—To determine the absorption of sulfanilamide when instilled into the conjunctival cul-de-sac, a 76 year old patient with absolute glaucoma was studied. The eye had an intraocular tension of 60 (Schiötz), with no perception of light. Paracentesis was indicated. A concentrated aqueous solution of sulfanilamide (0.8 per cent) was dropped in the eye as follows: Three drops were given every fifteen minutes for six times and then 3 drops every five minutes for six times before operation. The conjunctival cul-de-sac was thoroughly flushed three times with sterile distilled water before aqueous was withdrawn from the anterior chamber. Aqueous was obtained and tested for sulfanilamide. A faint trace of it was found but not in sufficient quantity to determine the amount—less than 0.1 mg. per hundred cubic centimeters.

The reason for the small amount of absorption of the drug after instillation into the cul-de-sac is that a sufficient quantity of the drug was not available in the cul-de-sac. One ounce (29.57 cc.) of a concentrated aqueous solution of sulfanilamide (0.8 per cent) contains approximately 3⅜₀ grains (0.23388 Gm.) of sulfanilamide. Hence, to obtain a dose by instillation in the eye equivalent to a single dose of 15 grains (0.972 Gm.) by mouth it would be necessary to have approximately 4⅛ ounces (123.228 cc.) of a concentrated solution of the drug available in the conjunctival cul-de-sac long enough for absorption.

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4. Lockwood, J. S.; Coburn, A. F., and Stokinger, H. E.: Mechanism of Action of Sulfanilamide, J. A. M. A. **111**:2259 (Dec. 17) 1938.

5. These determinations were made in the Harrison Surgical Research Department of the University of Pennsylvania by Miss Helen Lynch, through the courtesy of Dr. I. S. Ravdin, professor of surgical research.

6. Marshall, E. K., Jr.: Determination of Sulfanilamide in Blood and Urine, J. Biol. Chem. **122**:263 (Dec.) 1937.

7. Marshall, E. K., Jr., and Litchfield, J. T., Jr.: Determination of Sulfanilamide, Science **88**:85 (July 22) 1938.

Diffusion in the Aqueous After Ingestion.—Four days later the same patient was given 15 grains of sulfanilamide by mouth at 6 a. m. and at 12 noon. Aqueous was withdrawn from the anterior chamber four and a half hours after the last dose of sulfanilamide. The concentration of free sulfanilamide in the aqueous was found to be 2 mg. per hundred cubic centimeters of aqueous.

Diffusion in the Vitreous After Ingestion.—After an interval of five days, 15 grains of sulfanilamide was given orally to the same patient every two hours for three doses. Two hours after the last dose was given, liquid vitreous was withdrawn with a syringe and needle by puncture of the sclera, and a specimen of blood was taken for the determination of sulfanilamide in the blood plasma. The vitreous showed 1.5 mg. and the blood plasma 5.1 mg. of free sulfanilamide per hundred cubic centimeters of vitreous and blood, respectively.

Another investigation was made to determine the concentration of sulfanilamide in the vitreous and blood plasma of a patient aged 71. The eye was affected with phthisis bulbi. Fifteen grains of sulfanilamide was given orally every four hours for three doses. One hour and forty-five minutes after the last dose of sulfanilamide, vitreous and a specimen of blood were obtained. On analysis the vitreous showed 3.2 mg. and the blood 3.7 mg. of free sulfanilamide per hundred cubic centimeters of vitreous and blood, respectively.

Résumé of Investigations

Condition of Eye	Method of Administration	Amount of Sulfanilamide, Mg. per 100 Cc.		
		In Aqueous	In Vitreous	In Blood
Glaucoma	Saturated solution dropped in eye....	Trace		
Glaucoma	15 gr. every 6 hr.; total 30 gr.....	2		
Glaucoma	15 gr. every 2 hr.; total 45 gr.....	..	1.5	5.1
Phthisis bulbi	15 gr. every 4 hr.; total 45 gr.....	..	3.2	3.7
Phthisis bulbi	20 gr. in a single dose 32 min. before obtaining vitreous	..	1.1	

Rapidity of Diffusion to the Vitreous After Ingestion.—The determination of the rapidity of diffusion of sulfanilamide to the vitreous after administration by mouth was the last of the series of investigations made. The patient used for this study had phthisis bulbi of one eye. Twenty grains (1.3 Gm.) of sulfanilamide was given orally in a single dose. Thirty-two minutes after administration liquid vitreous was obtained with a syringe and needle. Analysis showed 1.1 mg. of free sulfanilamide per hundred cubic centimeters of vitreous.

CONCLUSIONS

Sulfanilamide was absorbed and found in the aqueous after local instillation into the conjunctival cul-de-sac.

The amount of sulfanilamide absorbed into the aqueous from the conjunctival cul-de-sac was less than 0.1 mg. per hundred cubic centimeters, whereas sulfanilamide was found in the aqueous and vitreous in a concentration of 1.5 to 3.2 mg. per hundred cubic centimeters after administration by mouth.

Sulfanilamide was found in the vitreous in a concentration of 1.1 mg. per hundred cubic centimeters thirty-two minutes after administration by mouth.

The ratio of the blood plasma concentration to the vitreous concentration could not be determined by these few tests.

The therapeutic value of sulfanilamide is known for infections caused by certain micro-organisms elsewhere in the body, and therefore because of its diffusion in the ocular fluids it should be considered equally valuable for the same infections in the eye.

The administration of sulfanilamide by mouth produces a much greater concentration of the drug in the ocular fluids than its local instillation in the conjunctival cul-de-sac.

Dr. Warren S. Reese, attending surgeon at the Wills Hospital, allowed me to use one of his patients for this investigation.

CLASSIC CHARACTERISTICS OF DEFECTS OF THE VISUAL FIELD

JOHN N. EVANS, M.D.
BROOKLYN

I. NEUROSCOTOMETRY

When the progress of a branch of medicine seems retarded, it is well to review the material at hand. New views on familiar subjects may open new avenues of activity. I shall review certain of these well known ideas with regard to defects of the visual field and will show how one can interpret such defects in a better way by consideration of factors often overlooked.

About twenty-five years ago perimetry (in its clinical application) began to expand rapidly, and in consequence there was much confusion for a while; studies of the peripheral field gradually became more reliable as a diagnostic aid, and the ophthalmologist began also to find that enlargements of the blindspot of Mariotte had great significance. Soon changes in the central field came into their own, and the ophthalmologist learned to recognize the classic defects of glaucoma, of retrobulbar neuritis and of certain degenerations. Then for a time students of the visual fields were led astray to wander up the blind canyon of color perimetry.

Throughout the entire period of this evolution there was much that was not understandable (as there always will be); but when the nutritional and vascular origin of some defects had been thoroughly demonstrated, a new light broke. It was possible to group great classes of defects according to the manner of their development.

Attention is thus focused on the fact that defects of the visual field can be classified as to origin under the headings of neurogenic defects, the neuroscotoma (studied by neuroscotometry), and angiogenic defects, the angioscotoma (studied by angioscotometry).

Before I review the basic evidence on which these hypotheses of origin are built, I wish to call to mind that the interpretation of defects of the central field depends on a great variety of data collected not only through ophthalmologic examinations but through general physical examination. No attempt should be made to interpret a chart of the visual field as a single entity; it can only be classified. It becomes the basis for certain assumptions as to the nature and site of a lesion.

The great complexities of the human organism lead to confusion if more than a minimum number of assumptions are made.

It would be helpful could I thoroughly review the present conceptions of the anatomic structure and physiologic function of the optic pathway and related structures. It is not the purpose of this paper to do so; in fact, such a mass of material—some disputable—would obscure the simple picture it is hoped to present.

NEUROGENIC DEFECTS OF THE VISUAL FIELD

The word "neurogenic" is coined to indicate that the defect of the field under consideration arises in association with a corresponding lesion of a fiber bundle of the retina or higher optic pathway. This does not mean that the defect arises from direct pressure on the fibers. It means only that the function of the fiber bundle is interfered with. It will be convenient to speak of the process, however, as though the disturbance is due to direct mechanical pressure.[1]

There are certain basic characteristics to which it would seem most ophthalmologists can subscribe. Thus, if the right occipital lobe is removed (say, on account of the presence of an abscess due to a foreign body, the optic radiation being completely divided), one can predict that the temporal half of the retina of the right eye and the nasal half of the retina of the left eye will be rendered blind. The corresponding fields—namely, the right nasal and the left temporal—will show half field defects.

If the foreign body does not cause the formation of an abscess but merely divides the upper outer part of the fibers of the optic radiations on the right side, most ophthalmologists will agree that there will be a partial field defect of each eye—a notching, or a quadrant defect.

As a third possibility, if a slowly expanding benign mass progressively impinges on the optic radiations of the right side, it is generally agreed that a notchlike defect will be initiated (as, placed above and laterally to the pathway) which will gradually become a quadrant and finally a half field, defect—hemianopia.

One might also find considerable agreement among ophthalmologists in explaining the evolution of the defects on the basis of this mechanism for the optic tracts.

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1. One might speculate that blind areas similar to defects of the fiber bundles might originate from lesions of the primary and cortical centers. In the present presentation I wish the reader to include depressed function of these centers as giving rise to true neurogenic defects. One can hope—though there is, at present, nothing to warrant it—that defects of this origin will eventually be recognizable by characteristic changes in the visual field.

This brings me to a consideration of the chiasm. Years ago Dr. Cushing and Dr. Walker stressed the point that a classic type of evolution in the field defects is associated with disease of the pituitary body. To be sure, they were not satisfied with the explanation that the defect arose directly from pressure of the tumor mass on the chiasm (and few accept so simple a concept). Nevertheless, if one considers the arrangement of the fiber bundles and the fact that the semidecussation (nerves, chiasm and tracts) straddles the region involved by the lesion (anterior in almost 80 per cent of normal decussations), one is able to predict the progress in a specific case with remarkable accuracy.

The diagram in figure 1 will amplify the point, though refinements of the distribution of the fiber bundles are omitted. In other words, the anatomic details make it seem unlikely that simple pressure at the chiasm can give rise to the classic field defect; yet the clinical findings demonstrate that the conception of direct pressure at the chiasm permits accurate interpretation for diagnosis and prognosis. Though doubtless a major factor, I make no reference to the blood supply of the pathway, as knowledge of the matter is inadequate.

So far there has been fairly general agreement as to the characteristics and predictability of defects of the visual field which arise through disturbances of the fiber bundles of the optic pathways. Explanation of these defects has been corroborated innumerable times by autopsy and at the operating table. There is one axiom, however, which has not been pointed out by other workers so far as I am able to ascertain:

The classic defect of fiber bundle origin is wedge shaped, with its apex pointing toward the fixation point. Its borders show no pronounced fluctuations,[2] and it corresponds to the arrangement of the fiber bundles of the optic pathway.

Consider hemianopia. As it evolves, it forms a wedge with its apex toward the fixation point; this becomes a quadrant (its apex toward the fixation point) and, finally, two quadrants (the half field defects) with their combined apexes still toward the fixation point. Further progress in the filling in of the next quadrant still maintains the apex toward the fixation point. Of course one must not expect this to be geometrically precise, but it is usual to such an extent that one can distinguish it from the defect incident to the advent of increased intracranial pressure. Recognition of this characteristic of defects of the fiber bundles is exceedingly useful in following the progress of a process. This will be emphasized in later paragraphs.

2. Transient variations, spontaneous or induced, appearing during the actual plotting of a defect.

EXPLANATION OF FIGURE 1

Fig. 1.—Diagram showing the evolution of the "chiasm field" and a posterior-superior view of the chiasm, elevated and distorted. One may assume that the distortion is due to an enlarging tumor of the pituitary body. The diagrammatic illustration is not designed to depict the anatomic arrangement of fiber bundles or the "pressure mechanism." Each circle represents the "zone of effect" of the increasing lesion. The changes in the field are assumed to arise from a combination of factors: (1) circulatory disturbances, (2) prying of bundle on bundle, (3) direct pressure of the mass and (4) possibly other factors.

When the disturbance has expanded to include circle *A*, the inferior nasal fibers are depressed in function and hence the inferior part of the retina, *A'*, from the nasal periphery all the way to the macula (the macula, *M*, and the papillomacular bundle have been "excised" to simplify the drawing). This means that the upper temporal visual field will show a "notching," or a quadrant defect *A''*. When this is complete there is a sharp dividing line between the temporal and the nasal half because the respective fiber bundles *A* and *D* at the chiasm are so widely separated as not to be involved by the lesions, but the dividing line of the upper and lower nasal quadrants may be less geometrically delineated, as the corresponding fiber bundles *A* and *B* in the chiasm are so closely related that dysfunction of one is likely to involve adjacent fibers of the other.

When the chiasmic disturbance has increased to circle *B*, the superior nasal portion of the retina, *B'*, is involved with a corresponding increase of the field defect from above downward into the lower temporal quadrant, *B''*. At this time there is a sharp dividing line through the fixation region. (It may divide, involve or spare the fixation point.) This stage of the defect may remain stationary for a relatively long time before the inferior nasal field shows involvement due to expansion into zone *C*, as it affects the superior temporal fibers. The last fibers, *D*, reached are those of the inferior temporal portion of the retina, *D'*, because they are laterally placed and tipped outward. Thus, the superior nasal field remains unaffected longest and with recovery is the first to show returned function, which then extends into the lower nasal, the lower temporal and finally the upper temporal quadrant, thereby reversing in recovery the order in which the quadrants were lost.

The inset depicts the true arrangement of fibers as seen from above (Wilbrand, H.: Der Faserverlauf durch das Chiasma und die intrakraniellen Sehnerven, Berlin, S. Karger (1929). The tracts are at the top of the illustration. The left optic nerve has been destroyed. One notes that the crossed fibers of the right nerve dip into the right tract and into the left optic nerve as they pass to the left tract.

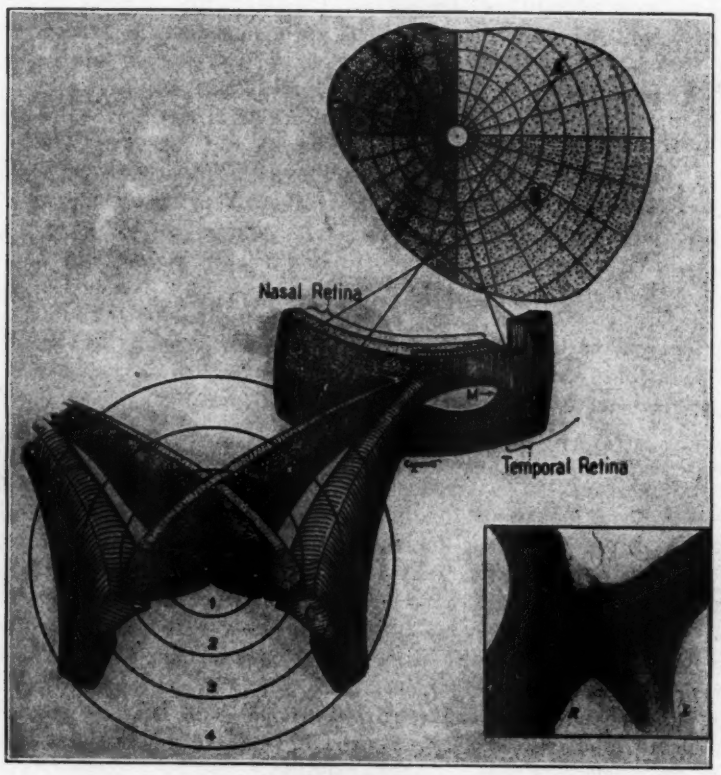

Figure 1

At this point I would remind the reader that the fibers from the retina temporal to the fixation point enter the temporal side of the optic nerve and follow backward in the temporal aspect of the pathway. One cannot be sure that they occupy the temporal half or one third or any particular fraction of the nerve; but it is known that complete division of these fibers gives a complete loss of function in that portion of the retina which is temporal to a vertical line through the macular line.

Thus far this analysis of acceptable evidence has progressed from behind forward to a series of so-called defects of the fiber bundles, the origin of which is debatable. I refer to those described as a typical accompaniment of glaucoma (and they occur often in other ocular conditions),[3] the enlarged blindspot, the Seidel sign, the Bjerrum sign and the Rönne nasal step.

Basic relations must be considered first.

The dividing line of hemianopias, as previously pointed out, passes vertically through the fixation point. However, the fibers from the

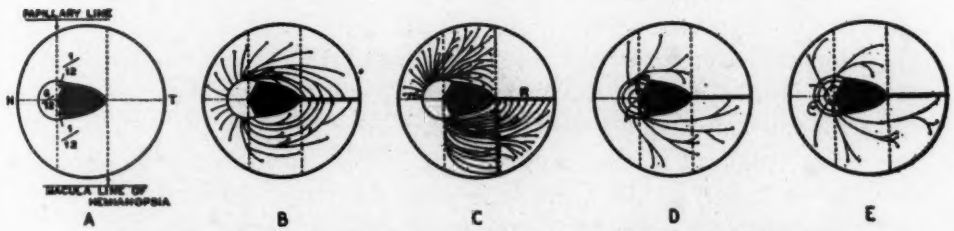

Fig. 2.—The topography of the arrangement of the fiber bundles in the retina. In *A* the shaded area represents the papillomacular bundle. The nerve head is divided into twelfths. Note the papillary and the macular line. *B* shows the classic diagram of the arrangement of the fiber bundles. One might wrongly conclude from this that when the crossed fibers in the chiasm are divided, the line of half blindness would pass through the blindspot. *C* shows that the fibers of the nasal half of the nerve are derived from all of the retina nasal to the macular line and that the fibers of the temporal half of the nerve are derived from the region of the retina temporal to the macular line. *D* shows the added conception that axially placed fibers in the nerve are derived from the region of the retina adjacent to the papilla and that fibers in the periphery of the nerve are derived from a more remote region of the retina, thus carrying out the plan of the Leber-Bunge-Fuchs theory. *E* shows the teachings of the opposing school (as compared to *D*), according to which the fibers in the axis of the nerve are said to arise from the retinal periphery and the fibers in the periphery of the nerve are said to arise from the region of the retina immediately adjacent to the papilla. Gradle refers to this as the Uhthoff-Wilbrand-Sanger theory. The diagram shows the nasal nerve fibers crossing the macular line. *PM* indicates papillomacular bundle; *R*, the horizontal raphe.

3. Even Bjerrum recognized that arteriosclerosis could give rise to the scimiter-like defect which he described.

nasal portion of the retina may be divided from the fibers from the temporal portion by an imaginary line through the papilla (the papillary line). This might lead one to expect that a lesion of the temporal fibers would render the retina blind from the temporal side of the nerve head to the extreme temporal periphery. Hence, the dividing line of temporal fiber blindness (hemianopia) would be through the blindspot, whereas it is known to be through the fixation point and to correspond to the vertical "macular line." It thus follows that the fibers in the nasal part of the nerve arise not only from all the retina nasal to the nerve but from all the temporal area of the retina up to the macula; thus, the nasal fibers in the nerve have to serve a great deal more retinal area than just that which is nasal to the papilla. This is more readily appreciated when one realizes that one third of the entire nerve head is reserved for the macula and that this one third is located in the temporal half of the nerve. This leaves a small area of nerve head for the fibers supplying the region temporal to the macula.

In other words, if a surface view of the optic nerve head is divided into twelve segments, six of these will be in the nasal half and six in the temporal half; but of the six in the temporal half, four twelfths are reserved for the macula, leaving only one twelfth in the upper part of the temporal half and one twelfth in the lower part for the region of the retina temporal to the fixation point. This crowding of the fibers into the nasal half of the nerve is so great that it can be seen with the ophthalmoscope as a blurring of the nasal edge of the disk.

It is next necessary that the modern conceptions of the arrangements of the nerve fibers in the retina be referred to. Ophthalmoscopic studies of the (anomalous) retinal medullated nerve fibers and of normal and of pathologic nonmedullated nerve fibers by red-free light have made possible the drawing of a pattern of the topography of the nerve fibers of the retina which is generally accepted.

According to descriptions of the classic arrangement, the fibers radiate in all directions from the nasal side of the nerve head into the retina; but from the temporal side of the nerve head there is the great ovoid fasciculus of the papillomacular bundle passing from the central third of the optic disk to the macula. Arching above and below this band, the fibers pass to the temporal part of the nerve from the temporal portion of the retina but to neither fasciculus crossing the horizontal raphe.[4]

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4. The word raphe is confusing. One must not compare it to other anatomic raphae. There is no dense line of compact tissue for the insertion of structures. It is a terminal line of end organs. A similar line of terminal end organs must also exist on the macular line, else there would be no vertical demarcation in the hemianopias. I am confident that the nasal fibers do not cross the horizontal

(Footnote continued on next page)

It has been seen that these fibers from the temporal half of the nerve (the papillomacular bundle is excluded from the discussion) can supply only the region of the retina temporal to the macula. The region above and below the papillomacular fasciculus must be supplied only by the nasal fibers of the nerve head (they terminate at the hemianopia line). In order to include this evidence, the classic picture of the arrangement of the retinal fibers must be modified.

Years ago when it became evident that examinations of the visual fields could be applied to clinical study, it became necessary to explain peripheral contractions, enlargements of the blindspot and central scotomas. Those whose attention was concentrated on the fiber bundles (I shall call them the neuroscotometrists) became divided into two groups.

One school (Leber, Bunge and Fuchs) declared that their studies made it evident that those nerve fibers which were located in the periphery of the optic nerve head served the region of the retina immediately adjacent to the nerve (periphery to the peripapillary area). Hence, damage to these would cause enlargement of the blindspot. Fibers which were located in the axis of the optic nerve head (adjacent to the vessels) served the peripheral portion of the retina (axis to the periphery). When these fibers were damaged, they contended, peripheral defects occurred.

The opposing school (Uhthoff, Wilbrand and Sanger) maintained that evidence which they had collected tended to show quite the contrary plan; that is, that fibers in the periphery of the papilla served the periphery of the retina (periphery to periphery), whereas the fibers placed in the axis of the nerve served the region of the retina adjacent to the nerve (axis to the peripapillary area).

Both schools accepted the idea that damage to the macular fibers caused a central scotoma.

Both schools had made elaborate studies and considered anatomic, physiologic and clinical material.

Irrespective of which arrangement one accepts as a working hypothesis, it is obviously necessary to make provision whereby the

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meridian of the retina, yet their low arching and lineal termination of end organs (rods or cones) produces no so-called raphe. Since the fibers do not cross, one would expect that the horizontal defect would be bound to occur from causes similar to those giving rise to the Rönne nasal step. The excessive crowding of fiber bundles of the nasal nerve head may explain this lack of horizontal defect in the temporal field. Thus, if a flaring bundle of fibers above the horizontal meridian and another below were overlapped by a third bundle, pinching in the horizontal meridian would always produce a wedge-shaped defect with a slanting upper or lower edge and never a horizontal edge. This seems to be the actual finding. One must still recognize the horizontal arrangement of the end organs in altitudinal hemianopias.

fibers in the nasal part of the nerve can pass over, under or between the temporally placed fibers in order to reach that area of retina which lies between the papilla and the macula. By any plan, some bundles will become intimately related to the "sharp" and resistant edge of the scleral foramen.

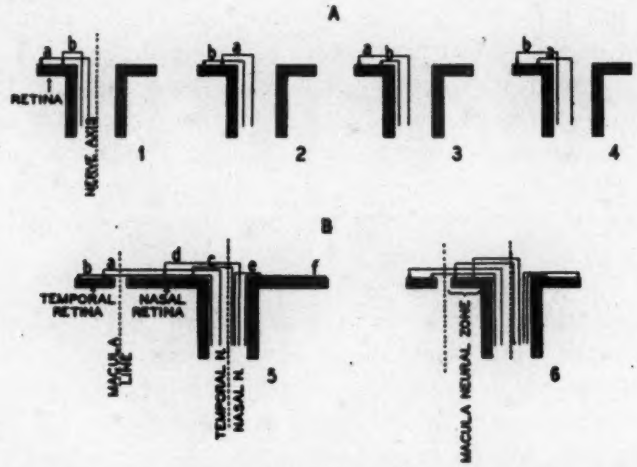

Fig. 3.—Diagrams of the possible relation of the distribution of the fibers of the optic nerve and retina. *A* shows the classic manner of indicating fibers, according to which none cross the axis of the nerve. In diagram *1* the fibers near the axis of the nerve are from the periphery of the retina, as shown by (*a*); those near the periphery of the nerve are from the region of the retina nearest the papilla (*b*). The fibers cross in the nerve and again in the retina. In diagram *2* the fibers near the axis of the nerve are from the region of the retina nearest the papilla (*a*) and those near the periphery of the nerve are from the region of the retina near its periphery (*b*). In diagram *3* the relation is the same as in diagram *1*, but there is no crossing of fibers. In diagram *4* the relation is the same as in diagram *2*, but fibers cross in the nerve. It is equally obvious that any of the crossings may occur over the edge of the scleral ring (retino-papillary junction). There is no reason why one may not assume that some or all of these patterns may be combined or exist separately in various segments. There may be great individual variation.

B presents the suggested method to show the fibers of the nasal half of the nerve must cross from the temporal portion of the retina, traversing the zone between the papilla and the macula. In diagram *5* the fibers near the temporal periphery of the nerve are from the temporal periphery of the retina and cross the macular line (*a*); those near the temporal side of the axis of the nerves are from the temporal central region of the retina and cross the macular line (*b*); those near the nasal side of the axis of the nerve are from the region of the retina adjacent to the nerve (*c*); those near the nasal periphery of the nerve are from the region of the retina nasal to the macula (*d*); those near the nasal side of the axis of the nerve are from the periphery of the retina nasal to the nerve (*e*), and those near the nasal periphery of the nerve are from the periphery of the retina nasal to the nerve (*f*). In diagram *6* an alternate arrangement of diagrams *3* and *4* are shown, including the conception of the nasal fiber supply to the maculoneural retinal zone (between the macula and the papilla).

Thus far it will be noted that in speaking of the neurogenic defects reference has been made only to the conductive elements—the nerve fibers. Many workers have felt that an attempt should be made to arrive at an understanding of the pattern of termination of the individual fibers. Careful studies have been made, but little progress has resulted. The one point of agreement seems to be that for the more central region of the retina a single fiber serves a single cone, whereas more eccentrically a number of rods (or cones) are served by a single fiber.

Figure 4 will help to show a few possible plans of termination, and it will be noted that a variety of shapes (scotomas) in field defects are possible when a group of fibers is damaged, depending on the pattern of termination.

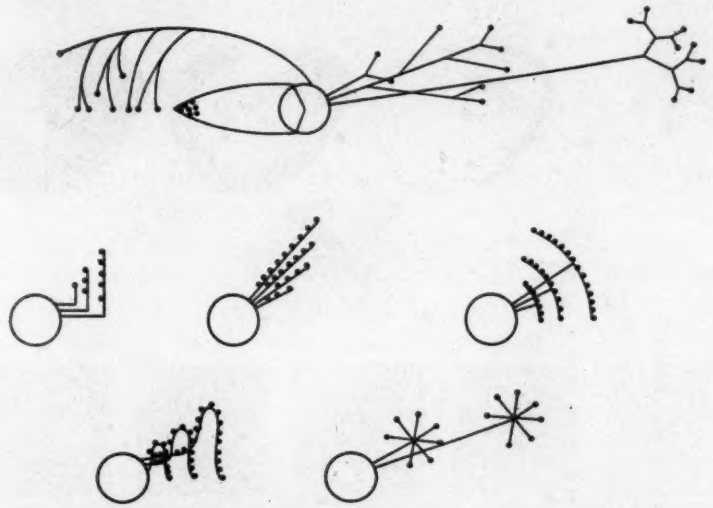

Fig. 4.—Diagrams devised to illustrate a few of the possible patterns of termination. It is probable that the uppermost pattern most nearly fits what little evidence is available. If the indicated fibers are divided, one will be able to visualize the effect that the pattern of termination will have in determining the shape, position and relations of the resulting field defects. In each instance except the last the division of all the fibers indicated will result in a wedge-shaped defect, but the division of a restricted number of fibers in one of the patterns will result in a radically different form of scotoma.

EVOLUTION OF THE CHANGES IN THE VISUAL FIELD IN GLAUCOMA

Before discussing the origin of the field defects of glaucoma, it is essential to review the classic evolutions through which they are said to pass. I shall phrase the description in the language of the neuro-scotometrist.

One must assume an elastic state of mind, as it is necessary to think in terms of retinal anatomy and physiology and at the same instant to transfer the mental picture to the rapidly changing plotted field.

Attention is first directed to the normal scotoma of Mariotte. At the earliest stage there is a vertical enlargement of the blindspot due to the appearance of a pointed defect from above or from below or both. This is the Seidel sign and is at present supposed to be the first field defect to appear. It may disappear and reappear from time to time, and by some it is said to occur slightly separated from the blindspot. When this elongation has become established, there is a suggestion of generalized enlargement of the blindspot and sometimes a slight decrease in the limits of the peripheral field.

The "sickle" defect of Seidel is said to arise by pinching and bending of the nerve fibers as they pass over the sharp rim of the scleral foramen incident to retreat of the thin lamina cribrosa due to

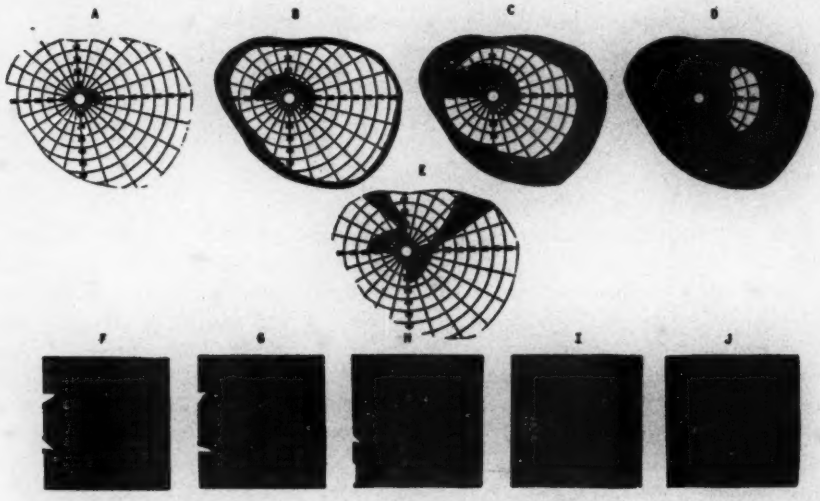

Fig. 5.—Field charts presented as idealistic representations of the evolution of the defects of glaucoma, for a complete elucidation of which see the text. *A* to *D* represent the conventional patterns according to their evolution by the neurogenic hypothesis, except that in many instances the neurogenic field should appear as it does in *E*, wherein the macular line would interrupt the scimiter-like and wedge-shaped defects. *F* to *J* show the evolution according to the angiogenic hypothesis. Though every attempt was made to stick to an idealistic pattern according to the hypothesis, these charts (*F* to *J*) simulate almost identically those found in any actual case which one might choose to select. This is different from the experience which one has in selecting charts from one's office records to illustrate the geometric patterns postulated by the neurogenic hypothesis.

the rise of intraocular pressure. One is asked to imagine that the pressure is rising continuously, so that it gradually involves first those fibers in contact with the scleral ring and later the more superficially placed fibers of the nerve head as they pass over the underlying bundles. The more superficially placed fibers are thus protected by the inter-

position of the deeper layers. The papillomacular bundle is said to be particularly resistant to this pressure, but the fibers which pass from the nerve immediately above and below it are either more vulnerable because of intimate relation to the scleral rim or they are specifically more sensitive.

At all events, there are extensions of the prongs from the top or bottom of the blindspot, so that the defect proceeds in a curve above or below and adjacent to the papillomacular bundle. These arching fibers terminate at the horizontal raphe, so that the full defect spreads out like the fiber fanning and ends abruptly in scimiter-like shape, its edge on the horizontal raphe. This is the Bjerrum sign.

Again it is said that at times the defect is separated from the blindspot and may grow both toward it and away from it. With the evolution of this sign one must realize that the fibers supplying the periphery and those supplying the retinal area about the papilla are also suffering, so that there is a gradual enlargement of the blindspot and also a decrease in the peripheral field. The nasal field is already partly occupied by the enlarging scimiter-like defect, so that a time eventually comes when the contracting periphery of the nasal field and the enlarging Bjerrum sign of the nasal field meet. For a reason not explained by the neuroscotometrists the Bjerrum sign usually occupies the upper field. This means that most of the upper nasal quadrant is obliterated, but the lower nasal quadrant has only been decreased in its periphery. The sharp horizontal edge of the fully developed Bjerrum sign hangs down, as it were, to make a step of blindness where the nasal quadrants meet. This is the Rönne nasal step.

With advance of the process, the sharply defined borders of this defect are lost, so that the field in general is oval, flattened from above and more restricted nasally. This is the loss of the nasal field described by the early workers. They placed it more peripherally because their objects were larger than those advocated today.

When one sees the almost geometric pattern of arrangement of the retinal nerve fibers, one is led to assume that the defects would be geometric in appearance. The appended diagrammatic illustrations should be compared with the charts mapped in actual cases. One must keep in mind that the increments of this evolution may be combined or blended in any given case.

The foregoing classic description seems logical and satisfactory until one realizes that one must include the material as summarized in earlier paragraphs.

Compression of the temporal fibers at the scleral opening is said to cause a corresponding crescent-shaped scotoma, which lies above or below the fixation point and far beyond into the region corresponding to the projected horizontal raphe, forming in the field the Bjerrum sign.

This explanation is, of course, incompatible with the statements made —that the fibers of the temporal half of the nerve serve only the region of the retina temporal to the macula. Pinching of the temporal fibers alone should cause an island of blindness in the nasal field limited centrally by a sharp vertical line, like that found in the hemianopias.

If the pinching of the fibers of the nasal half of the nerve occurred where they pass over the temporal lip of the scleral foramen, one would expect to find part of the scimiter-like defect ending in a sharp vertical line through the fixation point, as occurs in the hemianopias.

It is true that pinching of all the fibers, both from the nasal and the temporal half of the papilla, might cause the classic Bjerrum sign if the pressure was high and rapid enough in the onset. Opportunity to study the visual fields in cases of such fulminating lesions are of necessity rare. The usual onset is slow enough to follow the changes in the field through many cycles. If the temporal fibers really lie immediately against the scleral rim, they should show the effects of a rise of pressure first, so that at some stage there would be a partial scimiter-like defect with an abruptly delimited margin through the fixation point. Scotomas of this peculiar shape and location have not been consistently described. If the reader will start with the field as shown in figure 5 *E* and visualize an enlargement of the defects, he will develop a series of patterns which are certainly not the usual sequence in the evolution of the visual field met with in the majority of instances.

It is evident that the case for the fiber bundle origin of the defects of glaucoma is far from secure. There can be no justification for the exclusion of such acceptable data as have been collected for the explanation of defects of the optic pathway. One can further cite an infinite number of case studies which apparently refute the hypothesis of fiber bundle origin as applied to the interpretation of the field defects of glaucoma. Perhaps the most difficult defect to explain on the basis of the fiber bundles alone is the ring scotoma.

SITE OF LESIONS OF THE OPTIC PATHWAY CAUSING CLASSIC NEUROGENIC DEFECTS

It seems obvious that the greater number of those lesions which produce sector defects must arise from without the optic pathway and exert their influence on it; first, on its most peripheral nerve bundles, then on the deeper ones, the defect increasing as greater numbers of fibers are damaged.

Sector defects can arise from relatively rare tumors or vascular lesions from within the substance of the pathway when such structures or disturbances are inherent parts of the pathway (its own vessels, nerve

tissue or supporting tissue). Sector defects of this origin must be relatively rare, as the lesion would have to be limited to the external layers of the pathway.

Traquair has emphasized the fact that the fibers from corresponding retinal points of each retina are not "intimately paired" below the geniculate body but are above that center; hence the homonymous field defects of tract origin are not superimposable one on the other; they do not match. Supragenicular lesions—radiations and cortex—do produce fields which are superimposable, i. e., which are congruous.

Islands of blindness, however, would be most likely to originate within the substance of the pathway, centers or cortex. The most classic islands of blindness arising in this way are the scotomas of multiple sclerosis. Symmetrically placed islands are most likely to be cortical in origin, though they might arise from lesions in the neighborhood of the primary centers or elsewhere in the substance of the pathway.

Some one is bound to call attention to the classic defect of chorioretinitis juxtapapillaris. It is said to be due to the destruction of nerve fibers in the region of the retina adjacent to the nerve. Obviously this is so, as one can often see uncovered sclera at the site of the original lesion when atrophy has completely destroyed the retina and the choroid. A difficulty arises, however, when one finds only an island of blindness in the field when isolated areas of chorioretinitis occur in regions not adjacent to the nerve head. Such a lesion should also damage the radiating nerve fibers passing through it and hence give rise not to an island of blindness corresponding to the side of the lesion but to a wedge-shaped defect with its apex at the scotoma and its base in the periphery. As far as I know, fan-shaped isolated defects of the eccentric field have not been identified as classic for focal chorioretinitis other than the type occurring at the rim of the nerve head. These defects must, therefore, be explained by a mechanism other than simple destruction of fiber bundles.

One can readily see that the fiber bundles need not be considered in explaining the mere presence of defects, since sensitivity to light is an attribute of the rod and cone, not of the fiber. Thus, suspension of function of the rod and cone from local causes, such as may occur in glaucoma, can give rise to a great variety of field defects.

II. ANGIOSCOTOMETRY

Before the characteristic appearance of defects which arise as the result of vascular and nutritional disturbances can be considered, it is necessary to stress one or two points. The mere presence of the angioscotoma was not a discovery; it was evolved from little beginnings.

Numerous workers from the days of Mariotte on had delineated defects radiating from the blindspot.

The modern discovery that these shadows show pronounced variations under physiologic conditions and pathologic states provided a new and sensitive means of diagnosis particularly adapted to the recognition of early functional derangements.

Moreover, clinical angioscotometry in this modern sense has been securely established as a practical procedure by a host of workers.

With the renaissance of interest in this long known defect, useful scotoma data were bound to come to light.

When the modern maps were first presented it was necessary to demonstrate a relation of the bandlike shadows to the retinal vessel system; but even in those early papers an effort was made to emphasize the fact that the angioscotoma was not a map of the projected retinal

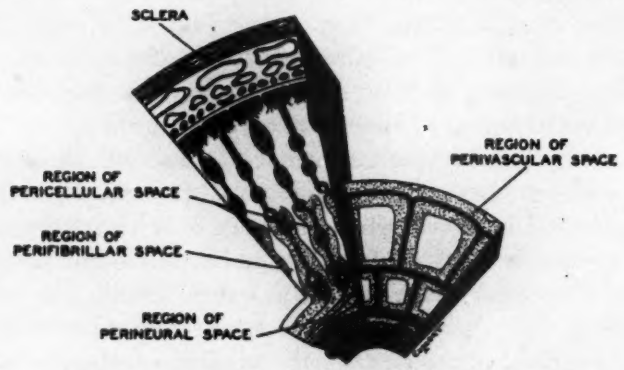

Fig. 6.—The retinal perivascular system.

vessels. The elaborate plottings were needed in the original presentations to establish the characteristics of the defect; but it is totally unnecessary to plot the entire tree in clinical examination. One needs only to study significant regions to obtain helpful information.

In reviewing the background of angioscotometry, I wish to recall the reasonably well established idea that each retinal vessel is surrounded by a perivascular space; that this is continuous with perifibrillar spaces about the nerve fibers and also with pericellular spaces. This system includes the synapses of the retina but is not directly related to the rod and cone neuron which gets its nourishment from the choroidal blood bed. The perivascular space is in intimate functional relation with the vitreous, and the space follows the large central artery and vein out through the papilla and empties into the orbital spaces. The perivascular space does not contain lymph but contains tissue fluid of vitreous and retina. It may even be accessible to cerebrospinal fluid.

One must recall that there are really three retinal vascular systems. The extreme periphery of the retina is supplied by a few twigs which do not primarily concern studies of the central field. The large vessels which pass from the nerve head, arching above and below the fovea, send spokelike projections inward, pointing toward the macula. The associated perivascular space system of the latter is of

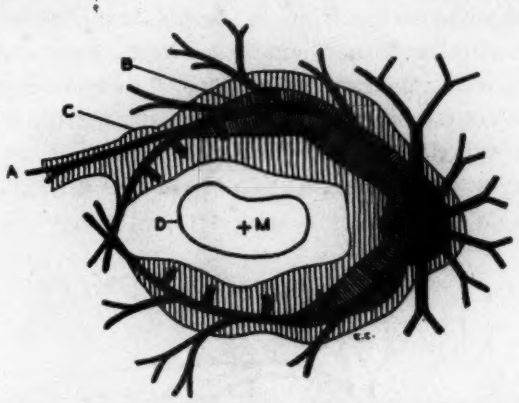

Fig. 7.—Progressive changes in basic angioscotoma. *A* shows a basic angioscotoma; *B*, widening in the earliest phase; *C*, an intermediate phase; *D*, the advanced widening wherein the individual branches have blended one into the other (the dipping of the upper border of this line represents the influence of gravity on the fluid of the inferior portion of the retina), and *M*, the macula.

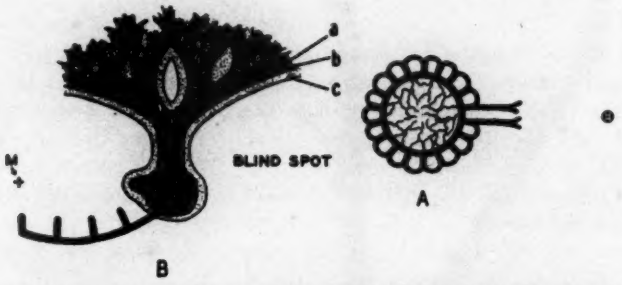

Fig. 8.—*A* shows the loops about the nerve head originating from the vascular circle of Zinn. The prongs extending from the left toward the circle and dot are the macular vessels shown as a single prong in *B*. This zone extends as a bridge when a macular lesion exists and gives rise to a cecocentral defect. *B* represents the wedge-shaped defect with its apex toward the blindspot, typical of the angioscotoma. The basic map of the normal angioscotoma is shown by *a*, the initial widening by *b* and the greatest widening by *c*. It will be noted that the widening of closely related branches blend one with the other toward the periphery, so that a solid area of blindness evolves associated with enlarging of the blindspot and a peninsular extension toward the macula (*M*).

great significance in the mechanism of the production of central scotomas.

The vascular circle of Zinn in the papilla sends radially, hooklike fingers or loops into adjacent retina.

There is also the system of dichotomous branching (see fig. 8*B*) which is seen with the ophthalmoscope and which forms two plexus-like ramifications deep in the retina, not reaching the rod and cone neuron.

The other system consists of a sort of vascular "raphe" extending from the temporal region of the macula to the periphery. It is formed by the small vascular twigs from the larger branches which arch above and below the papillomacular bundle. These twigs show slight interdigitations with each other in such a way that one can draw an imaginary line of sinuous course between them, from the macula to the periphery. This is the so-called "vascular horizontal raphe." One does not have to conceive of a geometric line which is not represented in clinical studies.

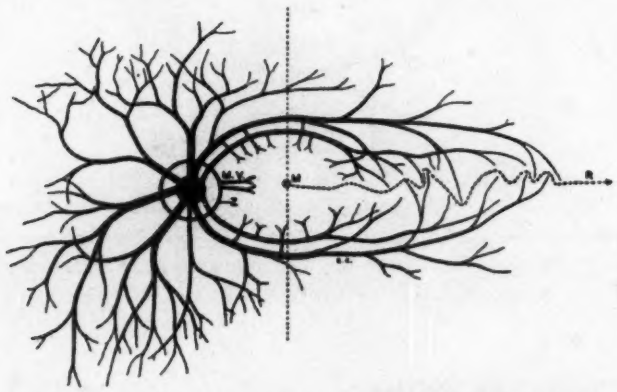

Fig. 9.—A diagrammatic representation of the retinal vessel tree showing the dichotomous branching. The zone about the blindspot is supplied by the vascular circle of Zinn (*Z*). Other points to note are the macular vessels (*M. V.*) and the macular line passing through the macula (*M*) and meeting the vascular raphe (*M-R*), which follows its sinuous course from the macula to the periphery between the superior and the inferior terminal twigs of the superior and inferior arching vessel systems.

Each of these vessel systems has its perivascular spaces. The knowledge of the anatomy seems reasonably secure.

The knowledge of physiology of the perivascular system is not so satisfactory.

There are cells which appear to have the power to dilate the perivascular space, thus lowering the pressure within it. Possibly there are constrictor cells which can reduce the diameter of the space. By dilation, the enclosed vessel can fill the space more completely than usual and hence raise the pressure within it. Likewise, constriction of the vessel should lower the fluid pressure in the space. Fluid passing into the

space from the vitreous should increase the pressure. Sudden drainage of this space should decrease the pressure within the space and the adjacent tissue. Obstructions to outflow should result in accumulation of fluid and thus develop a rise of pressure.

Escape of fluid might be obstructed by changes in the optic nerve head, rise of intracranial pressure or plugging of the space by inflammatory or waste products, tumor cells or hemorrhage. The escape of the fluid elements of the blood, as with obstructed venous return or circulatory inadequacy of any origin, is recognized as the classic cause of increased fluid content of the adjacent tissues; hence in these states the retinal perivascular spaces are overfilled.

Back pressure in the spaces produces edema of the synapses, thus shutting off the nerve impulses from the rods and cones. Obstructed nerve impulse lowers retinal response for the area affected, and the

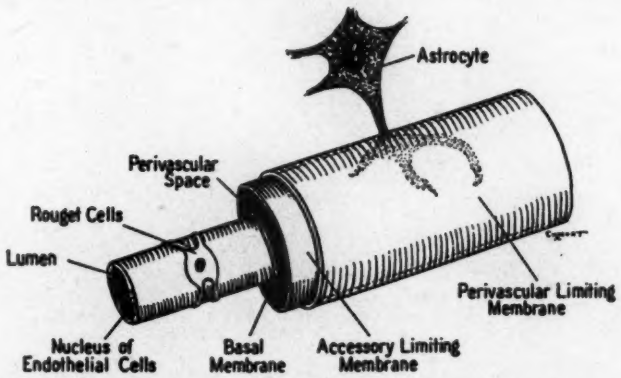

Fig. 10.—Diagrammatic representation of the structures of the perivascular space supplied to illustrate the postulated mechanism of widening and narrowing.

development of the corresponding field defect follows. Variations of the chemical composition of the fluid must obviously be felt by the retina in proportion to the degree of variation. Many data have accumulated to substantiate the hypothesis from which these statements are made. The literature should be consulted.

In order to comprehend the pathologic aspects of the problem, the variations which may occur as manifestations of physiologic processes must first be reviewed. No other method of clinical perimetry can follow such slight variations with the consistent fidelity of angioscotometry.

If the normal blindspot of Mariotte and one or two of the large angioscotomas emerging from it are plotted, a basic map is established, from which to note the variations.

Acceptance of the anatomic and physiologic conceptions as set forth in the preceding paragraphs gives rise to the question as to what

factors might be studied which could increase the passage of fluid from blood vessels and vitreous to and from the perivascular space. Thus, if a strong light is thrown into the subject's eye, the basic map becomes markedly widened; the same thing happens if venous stasis is produced within the eye by pressing on the jugular veins. Holding the breath, and even pressure on the opposite eye, produces a similar change. The same changes occur after physical exertion and a great variety of physiologic modifications. In every instance there is a widening of the basic defect when the process is sufficiently pronounced. The finer branches of the dichotomous tree blend one with the other, resulting in the production of wedge-shaped defect, its apex at the blindspot of Mariotte and its base in the periphery. Moreover, these defects show rapid variations in the course of a few minutes corresponding to those variations of the related physiologic processes which are known to characterize the vascular and the fluid mechanisms. This characteristic "transient fluctuation" of the defect is in strong contrast to the fixed character of the neurogenic defect. Thus, it would be entirely beyond one's expectation were the borders of a classic hemianopia to show transient advance or recession of a number of degrees with change of posture or holding the breath, all during the brief moments when such studies are in progress. It is, moreover, obvious that one can easily differentiate these fluctuating defects from the fixed neuroscotomas in a specific case by making digital pressure over the jugular veins. The borders of the angioscotoma will then immediately advance while the border of the neuroscotoma will remain unaffected.

When angioscotometry is applied to the study of pathologic conditions, it is found that there is no departure from the characteristics of physiologic defects except as regards extent.

The wedge-shaped defects obey the same laws. The vascular and nutritional changes of the retina are related not to their influence on the blood vessels primarily and not to any individual layer of the retina. They are related to the whole metabolic mechanism which controls the responsive functions of the retina. Decrease in these functions is reflected by concomitant modification of the angioscotoma.

It would seem logical to draw the following conclusion:

The classic defect of vascular origin is wedge shaped with its apex toward the blindspot of Mariotte. Its borders show transient fluctuations, and it corresponds to the patterns of the retinal vessel tree.

The progress of glaucoma simplex when followed by angioscotometry seems most rational if one adopts this conception:

Glaucoma is basically a manifestation of a deranged nutritive mechanism characterized by vascular crises and giving rise to a vicious cycle, terminating in such organic changes as venous thrombosis, angular synechia and retinal atrophy.

One's obvious endeavor is to make a diagnosis early, and since angioscotometry is particularly adapted to the early recognition of vascular manifestations its use is indicated in every study. By its aid variations can be recorded that are too minute for recognition by tonometry, ophthalmoscopy or other means. When one has outlined basic maps before the advent of the disease (fig. 5 *F* to *J*), one is provided with a normal standard for the patient. In the absence of a basic study in any particular case, a hypothetic standard derived from averages of a great number of cases is used. Routine studies are made of the angioscotomas arching above and below the blindspot and the small prongs extending toward the fixation point. As these widen with the progress of the disease, it is advantageous to study some of the radial defects. Owing to the influence of gravity on the circulation of the inferior portion of the retina, the angioscotoma arching above the fixation point is affected early, and its radial projections often dip toward the fixation point. When a large number of well controlled cases are studied, one finds defects which have been described in the literature as of fiber bundle origin but which cannot be accepted as such in the light of present ideas of anatomy and physiology as set forth in these pages. It seems evident, however, that there is probably some stage of the disease when compression of the fiber bundles will have involved the full depth of fibers passing over the scleral ring. At that time one would have the right to expect a field defect corresponding to a zone of dysfunction of the retinal fibers. Such a defect would, however, not alone explain all the destruction, because the vascular and nutritive derangements at so late a stage would be pronounced and of themselves give rise to extensive damage. There are doubtless times, however, when a remission of the circulatory insufficiency will make possible the recognition of damage to the fiber bundles. It is often possible to so modify the technic of examination as to permit the exclusion of minor vascular derangements and yet permit the plotting of classic injury to the fiber bundles. This is a useful plan to follow not only in cases of glaucoma but in cases of lesions of the optic nerve, such as syphilitic neuritis and tobacco-alcohol amblyopia.

Perhaps the best illustration of the relation of angioscotometry to neuroscotometry is demonstrated by a complete study of chorioretinitis juxtapapillaris. With the earliest signs of the lesion there is a localized vascular activity, followed by venous stasis, edema, exudation, migration of cells and finally atrophy. If the lesion is located at the upper pole of the papilla, there will be enlargement of the blindspot and widening of the angioscotoma below at the earliest stage. After a few hours the edema has increased to such an extent that gravity has carried it into the inferior region of the papilla, so that a pronounced wedge-shaped defect is found in the corresponding upper field. It is only in

the atrophic stage when the edema has disappeared that one finds the classic defect in the fiber bundles in the inferior field corresponding to the destroyed fanned out fibers entering the upper edge of the papilla. This neurogenic defect does not follow the classic characteristic of neuroscotometry.

One should always employ the technic appropriate to each method in a given case, and this is particularly valuable when intracranial lesions are present. The classic defects of the fiber bundles, such as hemianopias, are then distinguishable from the widened angioscotoma which arises with the onset of increased intracranial pressure. It is merely necessary to keep in mind the characteristics of these defects.

In reviewing the literature one can often recognize the onset of vascular damage by studying the charts presented to illustrate damage to the fiber bundles.

If the text supplies sufficient data, this interpretation is often substantiated. One must not get the idea, however, that it is always

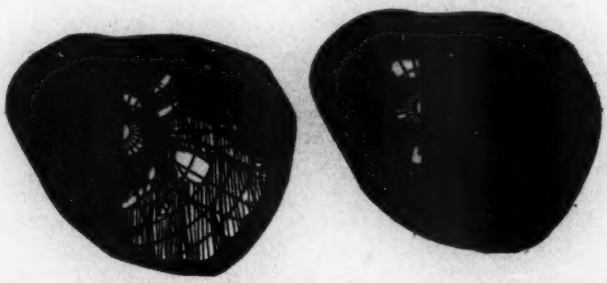

Fig. 11.—Diagrammatic representation of visual fields in which the hemianopia (indicated by the geometrically precise macular line) is distinguishable from the wedge-shaped defects which protrude from the periphery, the apexes of which in each instance point toward the blindspot and which have their origin in the angioscotoma. One might interpret this as part of the neurogenic defect, except for its characteristics, which when recognized would be interpreted as the advent of increased intracranial pressure. The chart on the left illustrates the appearance when the neurogenic defect occupies the nasal field, and that on the right illustrates the appearance when the neurogenic defect occupies the temporal field. After mapping the neurogenic defect on the perimeter, one then maps the angiogenic defect on the campimeter.

easy to distinguish the neuroscotoma from the angioscotoma even when all evidence is at hand.

TECHNIC

It should hardly seem necessary to point out that the technic of neuroscotometry is that of the universally acclaimed "quantitative perimetry." Though productive studies in angioscotometry are made by means of the tangent screen at 1 meter, the originally described technic is

much more satisfactory. One must use minute objects at close range with a columnating lens before the subject's eye.

To these simple statements as to technic, I wish to add one more, which is a common law to both and an essential at all times. "Move the object at right angles to the assumed border of the defect." One must have a mental picture of the fiber bundles of the optic pathway and of the retinal vessel tree.

SUMMARY

In the foregoing text I have tried to show that there are certain anatomic and physiologic principles which permit recognition of the characteristics of field defects and that there is a mass of material which has only served to complicate the interpretation. It has not been the object of this review to present new data but rather to insert the principle of neuroscotometry as a complement to angioscotometry for the better interpretation of clinical material. One can make better progress in the interpretation of defects if the following axioms are kept in mind:

1. The classic defect of fiber bundle origin is wedge shaped, with its apex pointing toward the fixation point. Its borders show no pronounced fluctuations, and it corresponds to the arrangement of the fiber bundle of the optic pathway.

2. The classic defect of vascular origin is wedge shaped, with its apex toward the blindspot of Mariotte. Its borders show transient fluctuations, and it corresponds to the patterns of the retinal vessel tree.

DIKTYOMA RETINAE

BERTHA A. KLIEN, M.D.
CHICAGO

E. Fuchs [1] in 1908 suggested the name diktyoma for a group of rare tumors arising from the pars ciliaris retinae, as their main characteristic is the production of membranes resembling the embryonic retina. Before 1908 4 instances of such tumors were reported. In chronologic sequence, the authors of these reports were Badal and Lagrange,[2] who classified the tumor as an adenocarcinoma; Emanuel,[3] who termed it a glioma of the pars ciliaris retinae; Verhoeff,[4] who gave it the rather appropriate name of teratoneuroma, and Ginsberg,[5] who called it a malignant epithelioma of the ciliary body.

Since then a total of 3 other cases has been added—one by Greeves,[6] a second by Velhagen [7] and the third by Böck.[8]

These tumors have the following features in common: (1) the young, usually infantile, age of the patient (the respective ages in the aforementioned reports were 8, 5½, 2½, 5, 1 and 16 years); (2) the flat growth of the tumor in the earlier stages, which tends to line the open surfaces rather than fill the cavities first; (3) the malignant character, which is shown sooner or later by infiltrating growth, and (4) the complexity of the histologic structure, which is already suggested by the varying terminology used by different authors and which will be discussed in detail in this report.

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7. Velhagen, C.: Gliomähnliche Geschwülste des Ziliarkörpers, Klin. Monatsbl. f. Augenh. **62**:571, 1919.

8. Böck, J.: Zur Kenntnis des Diktyoma retinae, Ztschr. f. Augenh. **69**:7, 1929.

In addition to these features, which were present in all the diktyomas, special characteristics in some of them were masses of neuroglia, demonstrated only by Verhoeff, Velhagen and Böck in the poorly nourished parts of the tumors, and islands of cartilage, found only by Böck.

REPORT OF A CASE

The following account is given because of the rarity of these tumors and because some hitherto unmentioned, unusual features were observed in this case.

C. D., a white boy aged 6 years, was brought to the clinic by his mother because of a white reflex from the left pupil, which she had noted for the first time one week prior to the visit. There had never been any attacks of pain or redness in this eye, and the child had never been severely ill.

External examination of the right eye gave negative results, and the fundus was normal.

Vision in the left eye was nil. The left bulb appeared to be of the same size as the right. The cornea was normal; the anterior chamber was slightly shallow, and the pupil measured 4.5 mm. in diameter and was derounded and fixed. Behind the lens there was a solid appearing, yellowish white mass. The intraocular tension was normal.

The eye was removed because of a suspected intraocular new growth.

The measurements of the enucleated bulb were: sagittal diameter, 23.5 mm.; frontal diameter, 23 mm., and vertical diameter, 23 mm. The eye was embedded in pyroxylin and sectioned from above downward. The superior calotte was also embedded and sectioned in a vertical direction, after it was evident that the base of the tumor was in the superior half of the eyeball.

Histologic examination showed the cornea to be normal, with a well developed anterior border ring in many sections. The anterior chamber was somewhat shallow; the iridocorneal angles were narrow but free, with only an occasional phagocyte in the meshes of the trabeculum corneosclerale. The surface pattern and stroma of the iris were normal. The lens appeared abnormally flat and measured 8.5 by 2.5 mm. in its largest diameters. Its posterior circumference and equatorial regions were surrounded by fibrous tissue, which continued forward on one side between the lens and the iris with a narrow extension. This fibrous tissue practically filled the posterior chamber and extended backward over most of the corona and orbiculus ciliaris of the middle and lower third of the eye. Behind the lens, completely filling the remnants of the cavum oculi, there was a tumor of peculiar appearance (fig. 1). The main mass was located in the upper half of the bulb and extended downward between the folds of the completely detached retina past the center of the pupil. The head of the optic nerve was drawn inward by the detached retina, and there was no retrodisplacement of the lamina cribrosa. The vitreous body was shrunken to a small mass and was completely surrounded by tumor; only posterior to it could the flat type of growth of the tumor still be demonstrated. Here the tumor crept along the inner surface of the retina, infiltrating its inner layers. Above this level of the tumor most of the retina was infiltrated and replaced by the growth or reduced to gliotic, degenerated masses.

The base of the tumor was visible only in the vertical sections through the superior calotte of the eye. The unpigmented ciliary epithelium became more and

more irregular toward the upper half of the eye, consisting in some places of from six to eight layers of elongated cells (fig. 2), but only at the base of the tumor was a direct transition from the unpigmented ciliary epithelium into the tumor demonstrable. Here, also, was the only place where the tumor had broken through the pigmented epithelium, infiltrating the stroma of the ciliary body.

The tumor consisted partly of tubules and bands composed of several rows of ill defined cells with oblong nuclei in a position perpendicular to the surface of the bands (fig. 3). Most of these bands had a distinct cytoplasmic seam on each side of the nuclear row, and in many of the tubules a delicate membrane resembling the external limiting membrane of the retina could be made out on the concave side. Between this membrane and the nuclear row there was usually a

Fig. 1.—General view of the tumor; horizontal section through its upper half. *V* indicates the vitreous body, and *F*, the flat growth of the tumor.

number of karyokinetic figures, a location of the mitotic process characteristic of the embryonic retina before its differentiation into layers, i. e., before the sixth week of life.

In other parts of the tumor, bands, loops and phantastic, spider-like figures of only one row of well defined, more cubical cells, of the appearance of the unpigmentary ciliary epithelium, prevailed. Narrow slitlike or large cystic spaces were enclosed within these convolutions and were filled partly with a pink-staining coagulum and partly with an extremely delicate fibrillary structure. Abrupt transition from the pleuricellular into the monocellular structures was observable in many places.

An unusual feature was the pigment content of some of these cells. The pigment was finely and evenly granular, located around the nucleus or between the nucleus and the convex side of the bands and tubules and present in the monocellular as well as in the pluricellular structures, one membrane often containing groups of pigmented cells side by side with the unpigmented ones.

Fig. 2.—Unpigmented ciliary epithelium, consisting of several layers of irregularly arranged cells.

Fig. 3.—Bands and tubules resembling embryonic retina. *K* indicates karyokinetic figures, and *T*, transition into low cubical epithelium.

In other parts the tumor consisted of solid cell conglomerations with rosette arrangement, as encountered in retinoblastomas (fig. 4). Here and there between these various structures were amorphous, necrotic masses containing tumor cells in all stages of degeneration.

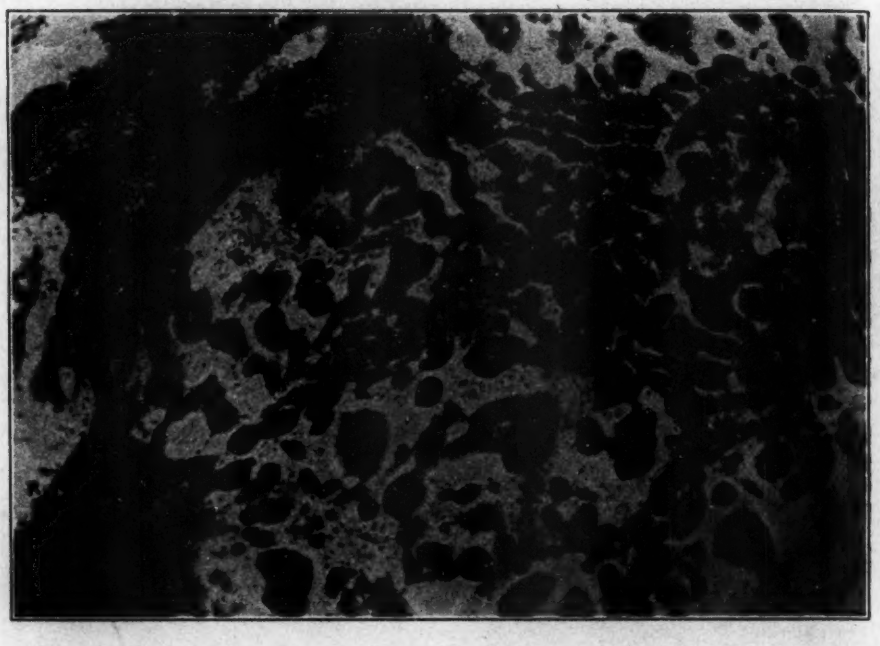

Fig. 4.—Solid cell conglomerations with rosette arrangement. *R* indicates the rosettes.

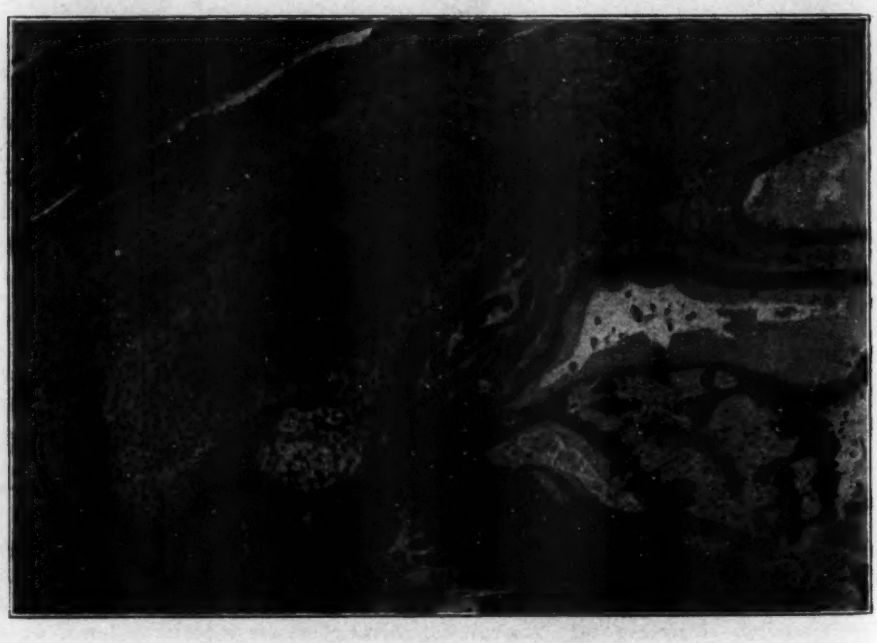

Fig. 5.—An island of cartilage.

While there was a complete lack of a reticulum between the tumor cells, a framework of connective tissue containing thin-walled blood vessels and numerous free hemorrhages could be traced between the convolutions of the tumor. Within this connective tissue scaffolding there were also irregular proliferations of the pigmented ciliary epithelium, more so near the base of the tumor, where it had been perforated and where its loose ends and fragments had become embedded partly in tumor and partly in connective tissue. Within extensive areas this connective tissue showed a peculiar, perhaps mucinous, degeneration, characterized by thick shreds of a more or less homogeneous material, which contained degenerated cells and stained an orange yellow with the van Gieson stain. There were also small islands of neuroglia in the posterior parts of the tumor, probably representing remnants of degenerated retina, but no extensive glial mass as an essential part of the tumor could be demonstrated.

Near the orbiculus ciliaris of one side, within the most solid portion of the tumor, there was an island of cartilage (fig. 5) surrounded by a distinct fibrous capsule.

An interesting feature was revealed by the ferrocyanide stain for iron-positive substances. In addition to phagocytes loaded with bluish granules of blood pigment, there occurred in some of the tumor cells neighboring free hemorrhages a diffuse bluish stain of the cell bodies, which in the tubular structures was deepest near the concave surface and which would indicate an absorptive activity, such as has been demonstrated by Meller [9] for the normal unpigmented ciliary epithelium.

COMMENT

In spite of the absence of secondary glaucoma at this stage, the tumor has to be pronounced malignant, as it had already destroyed a large portion of the retina by infiltrating growth and had begun to invade also the stroma of the ciliary body at its base. Böck's report of a diktyoma in a girl of 16 years, in whom external signs of an intraocular new growth were noted only six months prior to the enucleation, illustrates well that the clinical manifestations of the malignancy in these tumors may occur at a rather late date.

The histogenesis of the diktyomas seems to be clearly indicated by their histologic structure. The growth arises from the pars ciliaris retinae and is the counterpart of the retinoblastoma of the pars optica retinae. Only that portion of the secondary optic vesicle which later develops into unpigmented ciliary epithelium is capable of producing such a variety of cells, including embryonic retina.

Some changes in the tumor presented here are analogous to those in some microphthalmic eyes. Such is the presence of true cartilage, which may be explained by misplaced islands of chondroblasts, and such are the membranes and tubules of pigmented epithelium, which doubtlessly arose from an abnormal differentiation into pigment epithelium of the inner layer of the secondary optic vesicle; these call to mind the duplications of pigment epithelium demonstrated by von

9. Meller, J.: Ueber die Tätigkeit des Ziliarkörperepithels bei der Aufsaugung von Glaskörperblutungen, Ztschr. f. Augenh. **69**:113, 1929.

Hippel [10] in microphthalmic eyes. They are at the same time an illustration of the pluripotential qualities of the cells in this portion of the inner layer of the optic vesicle, which may produce, as in this tumor, cubical and cylindric epithelium, embryonic retina, cell conglomerations resembling those seen in retinoblastomas and in rare instances also pigmented epithelium.

Whether the malignant growth develops in a misplaced "anlage" or in unused, undifferentiated excess elements of the embryonic retina has been debated, but its origin as a congenital anomaly can scarcely be doubted, as large portions of the tumor retain the unquestionable characteristics of embryonic conditions.

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10. von Hippel, E., in Henke, F., and Lubarsch, O.: Handbuch der speciellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 2, p. 42.

PENETRATING INJURIES OF THE GLOBE FROM SPECTACLE GLASS

REPORT OF FOUR CASES

WARREN D. HORNER, M.D.
SAN FRANCISCO

Serious injury to the ocular globe from broken eyeglasses is not common, despite the enormous number of spectacles in every day use. I recently took care of such an injury which, peculiarly enough, is the second serious injury of this type that I recall having seen in twenty years. In mentioning the incident to some of my confreres, I was surprised to learn that three of them had encountered similar injuries within the five months previous to the writing of this report. I am reporting the series of 4 cases, 3 of them with the permission of these colleagues who have given me the necessary clinical synopses.

REPORT OF CASES

CASE 1.—Mrs. A. B., aged 30, seen by Dr. George S. Campion on Sept. 21, 1938, gave a history of being struck over the right eye, the blow shattering her rimless glasses. Examination in the hospital one hour later disclosed a linear penetrating wound of the right cornea 7 mm. long, extending obliquely from 3 to 11:30 o'clock. The sclera was uninjured. The anterior chamber was filled with blood. There was no prolapse of the iris, and the wound appeared sealed in good position. Perception and projection of light were normal. Surgical repair of the cornea was deemed unnecessary. Several small lacerations of the lids were sutured. The left eye was uninjured.

Treatment consisted of the use of atropine, the application of dressings and rest in bed.

On September 22 the anterior chamber was much clearer. A small torn bit of iris was visible together with lens débris from a traumatic cataract. The wound was closed. The patient did not have any pain. A roentgenogram did not show an opaque foreign body. Typhoid vaccine (5,000,000 organisms) was given intravenously as a precautionary measure. The maximum temperature was 39 C. (102.2 F.).

On September 23 fresh hemorrhage was filling the anterior chamber. There was increased pain, which was probably due to hypertension. The wound was still closed. Perception and projection of light were normal.

From the Division of Ophthalmology, Surgical Department, University of California Medical School.

On September 24 there was a small prolapse of the iris near the center of the wound. The chamber was still filled with blood. Projection of light was doubtful in the inferior nasal quadrant. The pain was less than on the preceding day.

On September 25 the prolapse was abscised. The edges of the wound were in good apposition. The hemorrhage was clearing. Projection of light was gone in both lower quadrants.

On September 29 the patient was discharged from the hospital. She rested in bed at home for two weeks. The wound healed but was slightly uneven. Projection of light was doubtful in all quadrants. The left eye was normal save for myopia of —3.50 D.

On October 15 slit lamp examination showed the corneal wound to be completely healed with small anterior synechiae. The anterior chamber was fairly clear but contained cells. About one third of the iris was obscured by clumps of lens cortex. The anterior vitreous was completely disorganized. The left eye was normal. There were no cells in the anterior chamber. Vision with correction was 20/20.

On October 19 the cornea of the right eye was steamy. The tension was 3 plus (on palpation), and there was no pain.

On October 27 a small piece of capsule (?) was seen in the nasal wound; the margin was cauterized with trichloroacetic acid.

On November 1 there were no cells in the anterior chamber. The tension was normal. There was no projection of light. The left eye was clear.

On Jan. 3, 1939, the tension was normal (on palpation). The eye was pale. There were no cells and no punctate keratitis. The anterior chamber was deep. The cortical material was clearing. A small remnant of iris was adherent to the lens substance. Perception of light was present but no projection. The left eye remained normal.

CASE 2.—Mrs. C. D., aged 52, seen by Dr. David O. Harrington on Aug. 6, 1938, stated that she had fallen down on a sidewalk the day before. A friend's version was that she was struck in the eye by a fist. Her glasses with metal rims were shattered, and the fragments penetrated the upper and lower lids of the left eye and the left eyeball. The patient was in a poor physical condition due to the excessive use of alcohol. While she was still in the hospital, delirium tremens developed.

Examination the first day was almost impossible because of her extreme nervousness, but on the second day, August 7, an estimate of her injuries could be made. There were several deep gashes in both the upper and the lower lid, particularly at the lower orbital margin. There was a jagged penetrating wound of the left globe. The wound crossed the major portion of the cornea from 2 to 8 o'clock and extended into the sclera for about 8 mm. The anterior chamber was filled with blood, and no details of the iris or lens could be seen. The patient was given typhoid vaccine (10,000,000 organisms) intravenously, which produced a rise in temperature to 100.2 F. with a secondary rise to 103 F. This precipitated the delirium tremens.

On August 10 two arterial silk sutures on arterial needles were placed in the scleral wound and one in the cornea. The prolapsed iris and ciliary tissue were abscised, and the scleral wound was covered by a conjunctival flap. The lacerations in the lids were probed for fragments of glass and sutured.

On August 12 the hemorrhage was absorbing nicely. The iris was seen through the upper part of the cornea.

On August 17 the upper part of the pupil was visible. A traumatic cataract was seen for the first time. Atropine, which had been administered previously, was continued. The hemorrhage continued to absorb.

On September 28 examination at the office showed normal vision in the right eye with correction. Projection of light was absent in the upper quadrants of the left eye. There was a completely mature whitish opacity of the lens. Slit lamp examination showed the lens reduced to about one-third normal thickness.

On November 21 there were no cells in the anterior chamber. The eye was very soft. Many large vessels were seen extending from the iris to the surface of the cataractous lens.

On November 21 the tension had risen somewhat, but the eye was still quite soft. Projection of light was absent in the upper quadrants but was fairly good elsewhere.

CASE 3.—Mr. E. F., aged 32, seen by Dr. Frederick C. Cordes on Oct. 31, 1938, was using an exerciser in a gymnasium when the handle on the end of the elastic snapped back, striking him in the right eye and breaking his rimless glasses.

Examination showed three linear corneal lacerations just outside the pupillary area. A fourth, penetrating, wound extended downward across the pupil to the 6 o'clock limbus. To this the iris was adherent at the lower pupillary margin. There was no evidence of injury to the lens and no retained spicules of glass. Treatment was instituted, consisting of the use of atropine and ethylmorphine hydrochloride, the application of a bandage and injections of omnadin (a nonspecific substance containing albumin, lipoid and fat).

On November 1 the atropine had broken the anterior synechiae. The anterior chamber was established. Many cells were present. There was moderate ciliary injection. Vision in the uninjured eye was normal.

On November 28 the eye was clear. There were no cells. The use of atropine was discontinued.

On December 16 an ointment containing 2 per cent quinine bisulfate was applied for the scars.

On Jan. 10, 1939, vision with correction was 0.6. The corneal scars were absorbing nicely. The eye was subjectively comfortable.

CASE 4.—W. W., an 18 year old Chinese boy, seen by me on Oct. 31, 1938, tripped over a rug in his room and fell flat on his face. The left lens of his rimless glasses broke, and the glass penetrated the left eye. He was seen at a municipal emergency hospital within an hour and was transferred to the ophthalmic ward of the San Francisco Hospital.

Examination showed a gaping wound of the nasal half of the left upper lid, extending horizontally for 10 mm. parallel to, and 6 mm. above, the margin of the lid, then turning upward and inward for 7 mm. more. The lid was penetrated throughout the greater part of the cut.

The globe showed a linear perforation of the sclera starting just above the upper limbus and extending nasalward for 6 mm., then angulating upward and inward for 5 mm. more. It was of the same shape as the wound in the lid. Vitreous exuded from the lips of the cut. The anterior chamber was about one-third filled with fresh blood. A fissure-like wound of the lens was seen at 11 o'clock. The globe was soft. Vision was limited to perception of hand movements at 2 feet (61 cm.). The right eye was uninjured.

The patient was given codeine and barbital and removed to the operating room.

Anesthesia was induced locally with a 4 per cent solution of procaine hydrochloride, and the lacerated upper lid was repaired with six silk sutures. The scleral wound was closed with four scleral and episcleral sutures of fine silk, Kalt scleral needles being used. Atropine was instilled, binocular patches were applied and typhoid vaccine (25,000,000 organisms) was administered intravenously. No frag-

ments of glass were found in the wound in the lid. The scleral wounds and the anterior chamber were carefully inspected for glass but were not probed. Roentgenograms were ordered for the detection of a foreign body.

Before the operation the boy's family was instructed to bring in the spectacles and all available particles of glass. The right lens was unbroken. It was a —2.25 sphere. The left lens, also a —2.25 sphere, was in four fragments, as shown in figure 1. Only one triangular piece, which was drilled for the nasal mounting, was absent. The missing piece measured 4 by 5 by 6 mm., roughly an equilateral triangle with obtuse angles. It probably was not concerned with the perforation.

On November 2 the dressing was changed. The lid was in good order. The anterior chamber was entirely filled with blood. The scleral wounds were closed. There was a moderate reaction, but the patient was in no pain. Roentgenograms were negative for an opaque foreign body.

On November 4 the patient experienced pain in the left eye. Typhoid vaccine (50,000,000 organisms) was given intravenously. The use of atropine was continued.

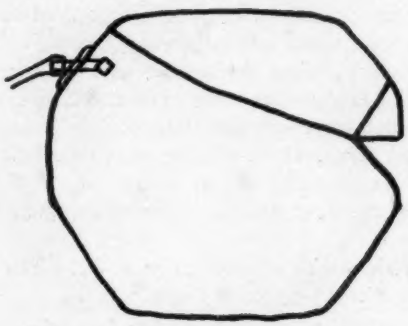


Fig. 1 (case 4).—Broken lens.

On November 5 the sutures in the lid were removed. The wound had healed. There was no increased irritation in the globe.

On November 7 the chamber was still filled with blood. The conjunctival chemosis was less. The scleral wounds were closed. The cornea was stippled. The patch was removed, and atropine and hot compresses were used. Perception and projection of light were present. The injection of typhoid vaccine was repeated.

On November 12 the anterior chamber was still filled with darkening blood. The scleral wounds were closed. The injection of typhoid vaccine was repeated.

On November 14 the cornea was assuming a greenish yellow color from blood staining. Typhoid vaccine (150,000,000 organisms) was injected.

On November 16 there was perception of light at 1 foot (30 cm.). Projection of light was uncertain in all fields. Tension (on palpation) was + 1. There was some increase in the circumcorneal injection.

On November 18 the scleral sutures had been cast off. The wound had healed smoothly. The cornea had a typical blood-stained appearance. Projection of light was doubtful in all quadrants.

On November 21 there was perception of light at 1 foot. Tension was normal. Slit lamp examination showed no reaction in the right eye. The patient was discharged from the hospital three weeks after the injury.

On Jan. 11, 1939, there was slight ciliary injection. The cornea had cleared in a concentric band 2 mm. wide save the area adjacent to the injured upper limbus.

Projection of light was poor nasally and below. It was present elsewhere. Vision in the right eye was 20/20.

On January 20 there was little injection. The patient felt no pain, and tension was normal. The cornea was blood stained within a concentric band 3 mm. from the limbus. Projection of light was still poor in the inferior nasal quadrant. There was perception of light at 1 foot. The right eye was normal on slit lamp examination.

COMMENT

Ocular injuries from broken spectacle lenses are fortunately rare, but they may assume grave proportions. Four cases of serious monocular injury are presented. Only 1 eye in 4 escaped with useful

Fig. 2.—Roentgenogram of glass spicules in a bowl of water.

vision (case 3; vision, 0.6). The other 3 are blind, with faulty or absent projection of light, and are candidates for any of the various late sequelae which may develop in old perforated globes. The final results are, therefore, problematic. As to etiology, two injuries were pure accidents. The injuries in case 2 and possibly in case 1 were tinctured with alcohol, so that one may say that spectacles and alcohol in excess constitute an unsafe mixture.

In none of the 4 eyes did panophthalmitis develop. All 4 patients received injections of foreign protein either at the first examination or as soon afterward as possible; 3 were given typhoid vaccine and 1 omnadin. I have long been convinced that this therapy, particularly

typhoid vaccine, should be routine in all accidental perforating wounds of the globe. My confreres apparently are of the same opinion.

It is interesting that blood staining of the cornea developed only in case 4. The anterior chamber was filled with blood for twelve days before the blood-stained appearance became evident. The chambers in cases 1 and 2 contained blood for five and eleven days, respectively, but the cornea did not stain. Secondary glaucoma did not occur in case 4, so that it was not a factor in the production of the blood-tinged appearance.

The question of retained fragments of glass in these cases is important. While one can, by probing, ascertain that wounds in the lid are

Fig. 3.—Glass spicule in the conjunctival sac.

free from glass, this is an unsafe procedure in the anterior chamber or the vitreous. The anterior chamber is usually so filled with blood that details cannot be seen. In such instances one must depend on roentgenograms. It was questionable to my mind whether particles of glass of small size would show on a film. To decide this point, I crushed an ordinary spectacle lens into small fragments. Six of these were selected and were rubbed on a carborundum hone to remove the sharp edges. They were then placed in a shallow aluminum bowl of water, and roentgenograms were made by Dr. R. R. Newell. All six of the fragments showed in the film (fig. 2). The seventh opaque dot was an artefact. The smallest two of the splinters of glass were then selected. One was dropped into the conjunctival sac of my own eye and the second into the eye of my associate, Dr. Malcolm Johnson, being preceded by a drop of solution of pontocaine hydrochloride. Roentgenograms were then

taken by Dr. Newell with the Sweet localizer. Both of the fragments could be seen on the plate. The smaller of these (fig. 3) measured only 1.5 by 1.5 by 2 mm. Whether a still smaller fragment would be visible, we did not determine. One may conclude, however, that a glass spicule which is as large as 1.5 by 1.5 by 2 mm. will show in an ordinary roentgenogram.

Minute foreign bodies, particularly glass or wood, in the anterior portion of the eye may be accurately demonstrated by the skeleton-free method of Vogt.[1] This method eliminates the shadows cast by the bones, thereby allowing softer rays to be used. It is described as follows:

> A small film-holder fitted with a piece of dental film is placed nasally to the eyeball and pressed as deeply as possible into the orbit. Thus the soft roentgen rays directed from the temporal side will reach the plate through the anterior part of the eyeball without interference by the bones of the orbit. By means of this method even the smallest particles can be accurately outlined.

This small series of cases indicates an already obvious conclusion, that rimless glasses are more liable to accidental breakage than those with rims. They seem also to be more dangerous, since 3 of the 4 injured patients wore the rimless type. Two of the three pairs of rimless lenses were drilled at 10 o'clock and 3 o'clock, as is usual nowadays. The drilling of the third pair is not known. It is my impression that this asymmetric drilling might favor irregular fragmentation with sharper spicules than the old 9 o'clock and 3 o'clock spacing, which favored splitting of the lens in half along the horizontal meridian. The fracture lines, of course, would depend mostly on how and where the breaking force was applied. It would appear that the latest type of rimless spectacles would be safer than either of the foregoing types, since the lenses are drilled only once for the nasal mounting and are backed up by the frame along their entire upper edge. To offset the hazard of injury through broken spectacle glass, safety lenses have been developed and are available at the better opticians on prescription. These incorporate a tough elastic transparent lamina between an inner and an outer glass plate, which prevents splintering should the lens be broken. As a proof of its effectiveness, one American company issues a certificate of insurance with each pair of its safety lenses, indemnifying the wearer up to $15,000 for any injury to the eye caused by splintering.

It is interesting, though not surprising, that 3 of the 4 injured patients wore myopic corrections. The fourth wore plus bifocal lenses.

490 Post Street.

1. Vogt, cited by Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 936. Wieser, St.: Mitteilungen über die skelettfreie Röntgenaufnahme des vorderen Bulbusabschnittes nach Prof. Dr. Vogt, Klin. Monatsbl. f. Augenh. **81**:234, 1928.

HISTORY OF A MARBLE BUST OF VON GRAEFE

RAYMOND L. PFEIFFER, M.D.
NEW YORK

Under the title of "Welche Bildnisse besitzen wir von Albrecht von Graefe," Prof. Dr. R. Greeff [1] published three papers in the *Archiv fur Ophthalmologie* in which he thoughtfully listed and described all the existing paintings, sculptures and photographs known to him of the

Marble bust of von Graefe.

father of ophthalmic surgery. The excellent bust of von Graefe at the Institute of Ophthalmology of the Presbyterian Hospital in New York, which has a most interesting history, was not mentioned, and I should like to record its existence.

1. Greeff, R.: Arch. f. Ophth. **138**:211, 1937; **138**:303 and 737, 1938.

This statue was given to Donders by his students and colleagues in 1872 on the twenty-fifth anniversary of his professional teaching at the University of Utrecht (Netherlands). It stood in his study with busts of Johannes Müller and Herman von Helmholtz, which were also given to him at the same time. At the death of Donders, these busts came into the possession of Frau Prof. Paul Krais, of Dresden, Germany, granddaughter of Donders. Later they fell to Frau Geheimrat Wilhelm Engelmann, of Berlin, Germany, whose husband had been a student of Donders and professor of physiology of the University of Berlin. More recently the bust was acquired by me from Madame Engelmann for the Institute of Ophthalmology and is now lodged in the office of the director of the institution.

The bust is life size, with the head leaning slightly to the right, and depicts von Graefe in the prime of life. It is of superb craftsmanship in Carrara marble and is attributed to Siemering, who did *das Denkmal* of the Charité-Krankenhauses in Berlin. The bust seems to be an exact counterpart of the head and shoulders of the famous and beautiful monument.

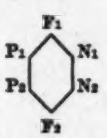
Clinical Notes

A MEMORY SCHEME FOR THE CARDINAL POINTS

JOSEPH I. PASCAL, M.D., NEW YORK

Every optical system has six important points, called cardinal points, associated with, and inherent in, the system. These are two principal focal points, F_1 and F_2, two principal points, P_1 and P_2, and two nodal points, N_1 and N_2. In the simplest optical system, such as a curved spherical mirror, these six cardinal points are reduced to three, as each pair of points coincide; i. e., F_1 coincides with F_2, P_1 with P_2 and N_1 with N_2. In a slightly more complex optical system, such as a single refracting surface, the six cardinal points are reduced to four, as two pairs of points coincide, i. e., P_1 with P_2 and N_1 with N_2. In a lens situated in air, the six cardinal points also are reduced to four but by a different kind of coincidence. Here P_1 coincides with N_1 and P_2 with N_2. In a complex optical system consisting of several refracting surfaces and several indexes, such as the eye for example, these six cardinal points are all separate and apart.

The two important distances in any optical system are the two principal focal lengths, measured from F_1 to P_1 and from F_2 to P_2, respectively. The two principal focal lengths are equal when the first medium (that is where the object is located or where the light starts) has the same index of refraction as the last medium (that is where the image is formed or where the light emerges). When the two indexes are unequal, the two principal focal lengths are in the ratio of these two indexes. There are, however, certain distances between specific cardinal points which are always equal to each other whether the system is of the simplest or of the most complex type. A knowledge of these relations is helpful in locating the cardinal points of an optical system and useful also in other ways. If the distances between any two points are represented by the two corresponding letters, $F_1\ P_1 = F_2\ N_2$; $F_1\ N_1 = F_2\ P_2$; $P_1\ P_2 = N_1\ N_2$ and $P_1\ N_1 = P_2\ N_2$. The first three are the important relations and can be easily memorized with the aid of the benzene ring, familiar to all doctors. F_1 and F_2, the two most important cardinal points, are placed at the top and bottom of the ring; the two principal points, P_1 and P_2, from which F_1 and F_2 are measured for focal length, are placed at one side, and the two nodal points, N_1 and N_2, are placed at the other side. Then the lines which are parallel to one another represent equal distances; that is, $F_1\ P_1 = F_2\ N_2$, $P_1\ P_2 = N_1\ N_2$ and



$F_1\ N_1 = F_2\ P_2$. If lines are drawn connecting P_1 with N_1 and P_2 with N_2, one gets two more parallel lines which represent equal distances,

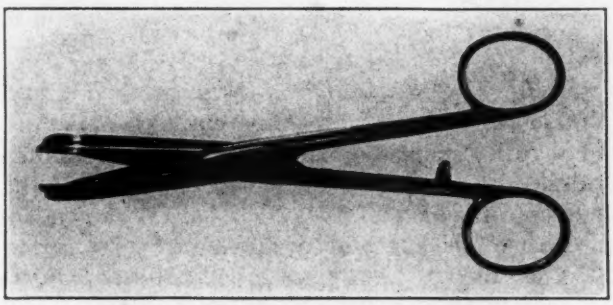
as $P_1 N_1 = P_2 N_2$. But as these lines are likely to complicate the simple benzene ring and as this equality can be obtained from the other three equations, it may be left out and only the three equations of the simple benzene ring remembered.

NEW ENUCLEATION SCISSORS COMBINED WITH HEMOSTAT BLADES

HUGO L. BAIR, M.D., ROCHESTER, MINN.

A frequently troublesome phase in the performance of enucleation is the control of hemorrhage after section of the optic nerve.

The commonest practice is to section the nerve with scissors, remove the globe, and then pack the socket until hemorrhage has ceased. This



Enucleation scissors combined with hemostat.

frequently prolongs the operation and does not always result in satisfactory hemostasis, particularly when a glass ball is to be inserted in Tenon's capsule.

Another practice is first to apply a small curved hemostat to the optic nerve well back toward the apex of the orbit, then to insert the scissors, the curve of which fits that of the hemostat, between the hemostat and the globe and thus sever the optic nerve. This prevents hemorrhage satisfactorily, but it is often difficult to manipulate the two separate instruments within the orbit. Frequently there is left only a short length of optic nerve attached to the globe, as in cases of intraocular tumor, in which a long length of nerve is desirable.

With these factors in mind, I designed enucleation scissors combined with hemostat blades on the convex sides of the blades of the scissors, as shown in the accompanying illustration. The blades of both the hemostat and the scissors are operated by the same handles, the former

From the Section on Ophthalmology, the Mayo Clinic.

closing slightly before the latter. With these scissors the proximal portion of the optic nerve is clamped and the section made by the blades of the scissors just distal to the point of clamping. The scissors are used in the usual manner, the rounded ends being first drawn across the optic nerve until the latter is well identified. The scissors are then opened, after one is certain the optic nerve is between the blades, and the blades are then pushed toward the apex of the orbit and closed. A single catch on the handles of the scissors holds them clamped in place. With the scissors left in place, the globe is removed, any necessary cutting of adhesions and other tissues being readily done with ordinary scissors. These scissors are manufactured by V. Mueller & Co. of Chicago.

AN IMPROVED TEST OBJECT FOR PERIMETRY

DAVID D. WAUGH, M.D., BROOKLYN

When motion is imparted to the test object in perimetry, a source of error is introduced. The motion of the object holder may be seen while the test object itself is invisible, and an inaccurate field is thus charted.

After seeing two instances of this error in various clinics, in which an immediate check revealed the discrepancy, I devised the following test object. It consists of a glass-headed pin on the end of a holder, the pin being bent at an angle of 90 degrees, 5 mm. from the center of the bead. When the handle is rotated between the fingers on its long axis, the bead will describe a circle of 1 cm. The bead will appear to the patient to be moving back and forth over a linear distance of 1 cm. The handle will appear stationary.

DILATATION OF CONJUNCTIVAL SAC IN ANOPHTHALMOS

Report of a Case

HENRY B. LEMERE, M.D., BEVERLY HILLS, CALIF.

Barbara E. was brought to the Marion Davies Clinic when she was 6 months old with anophthalmos on the left side (*A* of the accompanying illustration). The socket was extremely small, and there was no evidence of any globe. The child otherwise was perfect. There was no history of hereditary disease or of consanguinity. Gestation had been without any disturbing or toxic incident, even

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Presented before the Eye and Ear Section of the Los Angeles County Medical Association, March 27, 1939.

morning sickness being absent. Consultation of the literature and among my friends brought no suggestions for the improvement of this condition, and nothing was done for four years. After reading Paul Gougelman's article[1] I again got in touch with the parents and they brought the child in. The socket had little changed since I saw the patient when she was a baby (*B* of the illustration). It was impossible to introduce anything except extremely small objects into the conjunctival sac. The conjunctiva was healthy. There was no sign of microphthalmic globe and no movement of the conjunctiva on orientation of the right eye. The right eye was normal except for slight hyperopia. Small shells of increasing size were molded out of dental stent, and one about 7 mm. in length was introduced. During the following months the mother introduced larger and larger shells, till within three months the insertion of a small artificial eye was possible. In March 1938 an artificial eye was made (*C* of the illustration). At the time of writing

A, appearance of the patient at the age of 6 months; *B*, at the age of 4 years, and *C*, at the age of 5 years, after eight months' dilatation of the conjunctival sac and the insertion of an artificial eye.

the left interpalpebral fissure is still 3 mm. shorter than the right, and an oversize eye is being inserted at night to develop the socket further.

The procedure used is not new except for the use of molded dental stents,[2] but on account of my difficulty in getting any information as to procedure either from my friends or from the literature I think this case report may make the knowledge more available to others. If dilatation can be promptly started and an artificial eye introduced early, much mental anguish can be saved the parents.

1. Gougelman, P.: Fitting of Prostheses for Patients with Cryptophthalmos and Extreme Microphthalmos, Arch. Ophth. **18**:774 (Nov.) 1937.

2. Prijibylskaya, Y.: A Nonoperative Method of Dilatation of the Conjunctival Sac in Anophthalmos, Russk. j. opht. **13**:493 (May-June) 1931; abstracted, Arch. Ophth. **8**:608 (Oct.) 1932.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

ORIGIN OF THE VERTEBRATE EYE

GORDON L. WALLS, Sc.D.

DETROIT

When Froriep said, "Like Athena from the brow of Zeus springs the vertebrate eye into existence," he expressed the near despair which has been felt by many students of the problem of the origin of the vertebrate eye. The comparative anatomy of this intricate organ affords few clues to the history of the parts of the eyeball previous to their present association. The eye of the fish is as complex as that of man, and the structural variations of eyes are largely expressions of habit differences rather than stages in a majestic phylogenetic progress of increasing complexity and perfection, such as one sees in the heart, the brain, the ear or indeed in almost any other organ.

It is a commonplace to say of the retina that it is in reality a portion of the brain wall. The homologizations of the sclera and uvea with the dura and pia-arachnoid, respectively, follow naturally.[1] The lens can be plausibly explained on any of several bases. The noncellular humors have, of course, no ancestral homologues to be sought. The manufacture of a conjunctiva fixa from free skin of the head can be imagined between the lamprey and the shark and can be seen in the ontogeny of a salamander. The conversion of head myotomes into extraocular muscles to serve the eye on its attainment of eikonogenic capacity and the evolution of scattered cutaneous glands of the head into those compact ones peculiar to the ocular adnexa are processes which do not greatly tax the imagination.

Phylogenetically as well as functionally, the raison d'être of the entire globus oculi and its accessories is the retina itself. There must obviously have been a photosensory portion of the brain wall before there would or could have been an evagination and invagination thereof and a secondary conscription of dioptric and other accessories from among the convenient adjacent materials. The problem of the origin of the eye is thus essen-

From the Ophthalmic Research Laboratory, Wayne University College of Medicine.

1. However, the old concept of the sclera as a derivative of a hypothetic detached portion of the ancient chondrocranium—the "optic capsule"—is far from defunct in the textbooks, and no continuity of the choroid with the pial sheath of the optic nerve is demonstrable.

tially the problem of the past history of the retina. Since the establishment of the cell lineage of the retinal elements by Fürst in 1904, it has been clear that historically as well as ontogenetically the generative layer of the essential visual tissue is the so-called "neuroepithelium." The modern student therefore realizes that the real challenge to any theory of ocular origin is simply: Account for the rods and cones.

This was naturally not appreciated by the formulators of the earlier theories of ocular origin. The need for a review of this character lies in the fact that even the most recent of explanations have failed to take cognizance of a number of points regarding visual cells, the consideration of which leads rather to absolute rejection of most of the older theories than to their attempted inter-reconciliation, which is becoming more and more popular.

Lack of space and of any real necessity prohibits the consideration here of the various, mostly ill advised, derivations of the eye from the photoreceptors of subchordate animals.[2] The more distinctly defined hypotheses based on chordate morphology will be treated chronologically. Finally, an examination into the structural and developmental characteristics of vertebrate visual cells will afford an acid test applicable to those theories which are found to withstand criticism on other grounds.

MÜLLER'S THEORY—THE ANTERIOR PIGMENT SPOT OF AMPHIOXUS

Not quite a century ago a pigmented patch of epithelium was noticed in the anterior end of the neural tube of Amphioxus, the acraniate "grandfather of the vertebrates." On the basis of its pigmentation the anterior pigment spot (fig. 1, *aps*) was concluded almost at once to be an eye and drew the attention of many investigators, some of whom thought it to be normally paired and saw in it a lens, an optic nerve and even rods and cones.

In the first attempt ever made at a phylogenetic study of the vertebrate eye, W. Müller (1874) derived the latter from the anterior pigment spot and was quickly supported by Langerhans (1876). Kohl (1890) found no innervation and considered the spot possibly a thermoreceptor; but in the same year Ayers insisted that it was a paired directional eye exhibiting traces of higher retinal structure. Some early experiments indicated that the spot was indeed not photosensitive at all, and the work of Nagel (1896) and Hesse (1898) led to the same conclusion; but Edinger (1906) reaffirmed its innervation, and the idea persisted that it must be a sense organ of some sort. The refined experiments of Parker (1908*a*) and Crozier (1917) once more demonstrated its insensitivity to light, and Franz (1923) explained Edinger's nerve

2. The reader may profitably study Hagedoorn's (1936) ingenious derivation of the vertebrate eye from "phylogenetically antique" invertebrate prototypes of its various parts.

fibers as having connections elsewhere. As late as 1929, however, Pietschmann was still supporting the supposed innervation and visual nature of the spot. In the most recent work (1934) Wollenhaupt expressed agreement with Franz that the spot is not innervated and that its function is to cast a shadow on the photosensory "infundibular organ" (fig. 1, *inf*), thus making precise phototropic responses possible. The pigmented spot is not an eye but a part of a visual complex in which it plays the unique role of a negative of a lens.

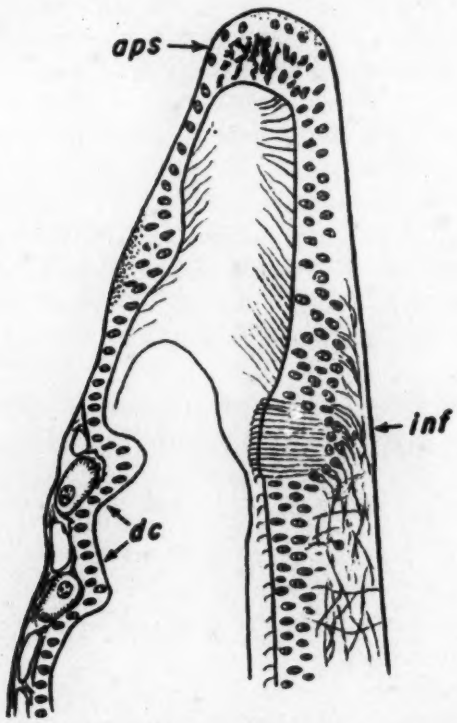

Fig. 1.—Sagittal section of the brain of Amphioxus, in the position it normally has in the living animal. The anterior pigment spot is indicated by *ops*, the infundibular organ by *inf* and two dorsal cells of Joseph by *dc*. (Redrawn, modified, from Franz, after Boeke.)

LANKESTER'S THEORY—THE EYE OF THE ASCIDIAN LARVA

In the "tadpole" larva of Ascidia the visual organ consists of a portion of the so-called sensory vesicle, attached to the brain and communicating therewith by a pore. It has a retina, pigment mass and dioptric body. The first descriptions of this eye were given by Kowalevsky (1871) and von Kupffer (1872) from dissections, and according to these authors the lens faces inward toward the brain, so that the retina, lying peripherad from it, can be stimulated only by light which

has traversed the transparent body of the animal from a mostly ventral direction.

In 1880 Lankester reasoned that the original vertebrate must have been transparent and had an eye or pair of eyes inside its brain "like that of the Ascidian tadpole." He imagined the eye-bearing portion of the brain being forced to move out toward the skin as the animal grew denser and opaque and thus accounted for the endoneural stage of the ontogeny of the vertebrate eye. Lankester did not suggest a homology or a genetic relation of the two types of eyes, but the "ascidian theory" is ordinarily attributed to him.

Balfour (1881) thought such a homology reasonable but considered it more likely that the ascidian eye had been derived from that of the vertebrate by degeneration. He pointed out that to hold the ascidian theory one would have to imagine the original lens being replaced by the

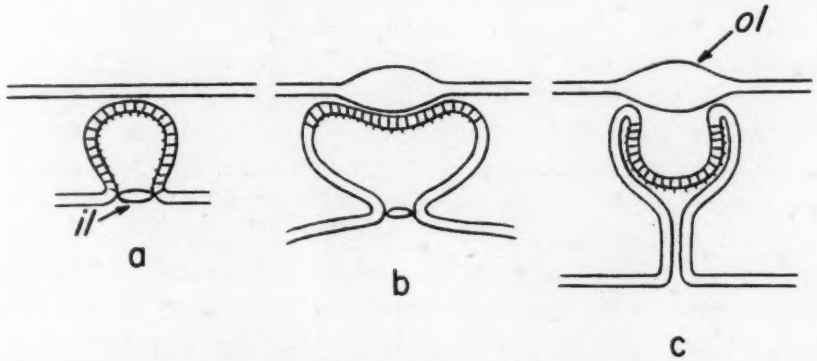

Fig. 2.—Drawings illustrating the ascidian theory in its original form. In *a*, *il* indicates the "inner lens" of the ascidian eye; in *c*, *ol* indicates the definitive "outer lens" of the vertebrate eye; *b* shows the hypothetic transitional stage. (Redrawn, modified, after Jelgersma.)

definitive vertebrate lens, with a phylogenetic stage at which two lenses (fig. 2 *b*) would be in use on opposite sides of the retina.[3]

This mental feat was not difficult for Jelgersma (1906), who evolved an elaborate ascidian theory (fig. 2) without knowledge of Lankester's and Balfour's statements.[4] Jelgersma considered the ascidian visual

3. Balfour seems not to have noticed that the same difficulty is offered to his interpretation of the ascidian eye as a derivative of the vertebrate organ. He perhaps imagined the eye to have lost its outer lens entirely and later to have recovered partially from its degeneration by producing an inner lens.

4. In fact, independently of Schimkewitsch (v. i.) Jelgersma explained the regenerated Triton lens as atavistic and comparable to the "retinal" lens of the ascidian eye. He and other proponents of the ascidian theory might have strengthened the latter considerably by borrowing Schimkewitsch's view, that the "inner (retinal) lens" must have become functionless before the "outer (definitive) lens" appeared.

organ to represent a stage in the development of the vertebrate eye, since it, like the primary optic vesicle, is globular and communicates with the neural canal. For this and other reasons he favored the ascidian hypothesis over the then recently announced theory of Boveri, which, Jelgersma pointed out, offers no hypothetic intermediate stage which is matched by an actual stage in vertebrate embryologic development.[5]

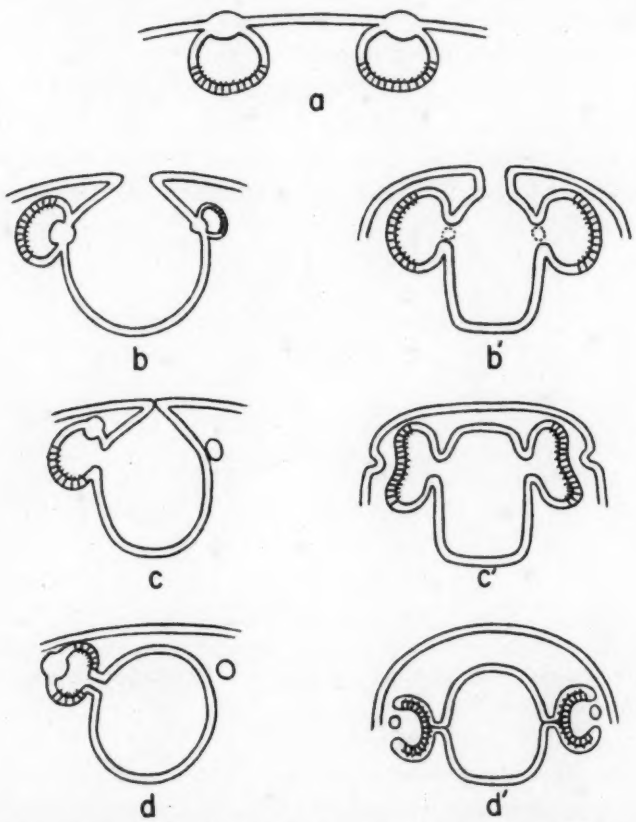

Fig. 3.—Drawings illustrating Froriep's derivation of the ascidian and vertebrate eye from ancestral superficial *Grubenaugen*. The stages in the evolution of the ascidian eyes, showing the degeneration of one member of the pair, are shown in *a*, *b*, *c* and *d*; the statocyst portion of the sensory vesicle is omitted for simplicity. The stages in the evolution of vertebrate lateral eyes are shown in *a*, *b'*, *c'*, and *d'*. (Original drawing.)

Froriep (1906 *a*) injected fresh information by showing that in sectioned material the ascidian eye is seen to be not median but located

5. But von Kupffer (1890) had claimed a median ventral anlage for the lateral eye of Petromyzon which would fit into Boveri's series of diagrams. Keibel (1928) found no such initial unpaired stage, however, and it is probable that von Kupffer had misidentified the embryonic infundibulum.

toward the right side. He interpreted a degenerate mass on the left as the vestige of a member of a former pair of eyes, only one of which remains. Froriep credited the ascidian theory with accounting for the origin of the vertebrate eye in a location (endoneurally) which in any present vertebrate could never be reached by sufficient light for vision; but he then proceeded to demolish the theory.

The ascidian lens is actually located peripherally in the sensory vesicle, and the functional retina lies on the brain side. Thus the eye looks directly outward and not through the entire thickness of the animal. The orientation of the retina with respect to the light source is thus the same as in most invertebrate eyes, not inverted as in the vertebrate.

Froriep considered the ascidian and the vertebrate eye to be homologous in that they have originated from a common ancestral structure, but he denied the possibility of the derivation of either from the other. The ancestral complex is, he stated, simulated by the *Sehgruben,* or foveolae opticae (fig. 6 *a, f*), of the vertebrate embryo. These represent ancient, superficial *Grubenaugen,* which were probably provided with lenses and which became carried inward in the evolving neural tube. The ascidian eye is thus more primitive than the vertebrate structure (since it retains the old lens [6]), but the *Sehgrube* stage of the latter approximates more closely the ancestral condition of both. On reaching the secondary endoneural location, according to Froriep, the ascidian eye regained efficiency by shifting the lens dorsolaterad and rearranging the sensory and pigmented portions to permit of vision in directions away from the brain (fig. 3). The vertebrate eye, on the other hand, has discarded the endoneural lens and obtained a new one, leaving the retina inverted.

Tretjakoff (1913) even denied the possibility that the vertebrate eye was originally vesicular and expressed the belief on ontogenetic grounds that it never possessed an intrinsic lens and that the eventual vesicular form was brought about mechanically by the ventrad growth of a hypothetic choroid plexus (fig. 4, *cp*) the outer leaf of which then expanded dorsolaterally to lay down the pigment epithelium. It is the umbracular remnant of this choroid plexus (fig. 4, *um*) at the dorsal lip of the pupil in lower vertebrates which is the usual source of the regenerated lens.[7] Tretjakoff's ingenious diagrams, however, have little

6. It also retains its pigmentation; but the foveolae opticae are pigmented in some forms (Froriep, 1906 *b*).

7. See footnote 4. One wonders why more critics of the ascidian theory have not asked, like Tretjakoff, why the "outer lens" should ever have evolved. If the regeneration of a lens from the pupillary margin, such as can occur in lower

(Footnote continued on next page)

in common with actual stages in ontogenetic development, and his explanations of the changes in relation are certainly not superior to those of Schimkewitsch, whom he criticized severely.

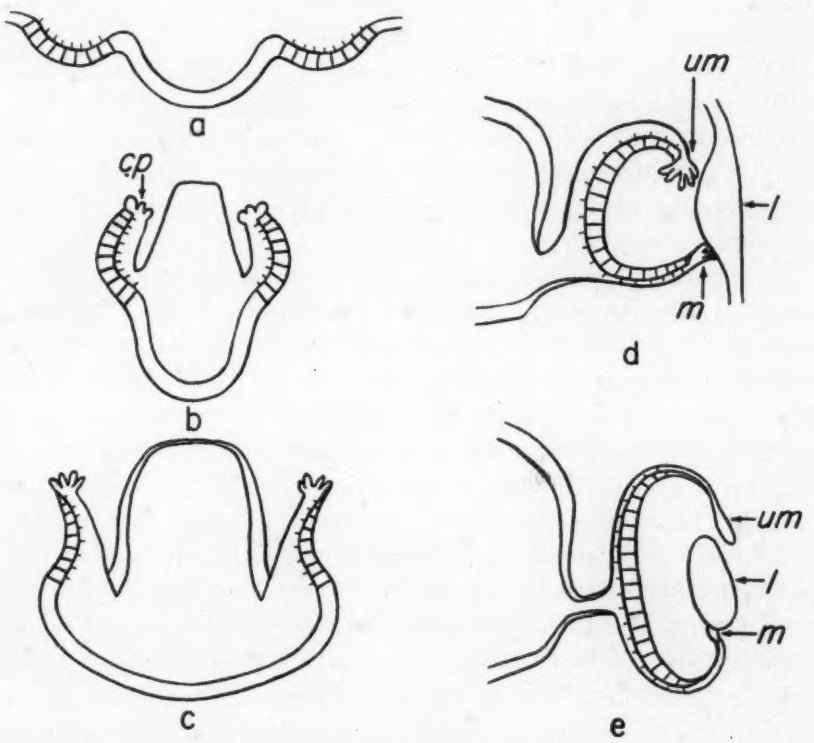

Fig. 4.—Drawings illustrating Tretjakoff's explanation of the origin of the invaginated optic cup, the regenerated lens and the lens. The foveolae opticae stage is shown in *a*. In *b*, *cp* indicates the hypothetic choroid plexus, which in *c* has grown ventrad and then expanded dorsad to create a two-layered cup by laying down the pigment epithelium. In *d* is shown the attachment, *m*, to the skin, which has evoked the lens, *l*; also the umbraculum, *um*, formed from the choroid plexus and corresponding to the pupillary nodules of amphibians. In *e* are shown the final condition with the retractor lentis muscle, *m*, attached to the free lens, *l*; and the umbracular remnant, *um*, from which a lens may be regenerated. (Redrawn, modified, after Tretjakoff.)

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vertebrates, is atavistic, it would seem that the eye need never have abandoned this "inner," or "retinal," lens. Schimkewitsch, to be sure, placed it in a useless location (fig. 12 *b*) in his 1902 diagram of the invagination stage of the hypothetic ancestral eye; but Tretjakoff has pointed out the many other locations of the regenerated lenses and lentoids besides this one and interpreted the regeneration as merely indicatory of the capacity of ectoderm everywhere to form lenses in the vicinity of the optic cup or even in the latter's own tissue.

BALFOUR'S THEORY—THE INTEGUMENTAL PHOTORECEPTORS

In 1881 Balfour proposed that the vertebrate retina once functioned as patches of photosensory dorsal ectoderm and that these were involuted when the neural tube evolved, coming to lie in the position of the primary optic vesicles (fig. 5). Thus the retina appears to derive from the brain but was not evolved there. The transparency of the pelagic ancestor permitted photoreception at all stages of the involution, but subsequent body growth necessitated an outpocketing toward the skin.

This theory accounted for the "anomalous" inversion of the retina as well as for its cerebral origin—great differences respectively from most invertebrate eyes and from other vertebrate sense organs. It was also advanced by von Kennel (1881) and was supported by Dohrn (1885), who had expressed the same notion, but less clearly, in 1875. Keibel (1906) and Franz (1934 *a*) also considered it certain that the

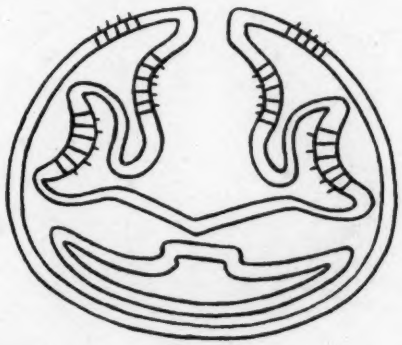

Fig. 5.—Drawing illustrating Balfour's theory. Patches of photosensory cells are shown in the successive positions which they are supposed to have occupied before and after the evolution of the neural tube and the optic evaginations therefrom. (Redrawn, modified, after Parker.)

vertebrate eye traces back to the skin. Jelgersma (1906) derived the ascidian eye from an integumental eye in this same fashion and to this extent favored Balfour's hypothesis. Froriep (1906 *a*), as has been seen (fig. 3), made use of Balfour's idea to link both the ascidian and the vertebrate eye to a common ancestral integumental eye, but with the embellishment of an assumptive lens for the latter. Balfour himself never carried his idea that far.

Froriep (1906 *b*) was also the first to lay great emphasis on the foveolae opticae, those paired pits which for years had been seen on the open cephalic plate in many sorts of embryos. These depressions, without active change, become the primary optic vesicles on the closure, over them, of the neural tube (fig. 6). Locy (1894) had already discovered two additional pairs of such foveolae in elasmobranch embryos and had

explained them as the anlages of the (originally paired) epiphysial eyes of the diencephalic region. Lange (1908), independently of Froriep and of Balfour, also interpreted the foveolae opticae as representing a phylogenetic stage in the evolution of the lateral eyes.

Schimkewitsch (1921)[8] elaborated Balfour's simple theory most extensively; but some of his ideas are more pertinent to later sections of this review and will be omitted here. Schimkewitsch considered the lateral eyes to have been originally superficial *Grubenaugen,* carried secondarily into the neural tube (fig. 7). They were merely the most anterior of a whole series of metameric pitlike sense organs, which had probably not always been sensory and only the first three or four pairs of which were photosensory. From the latter, which were perhaps all at first constructed like existing pineal eyes with a lens formed in

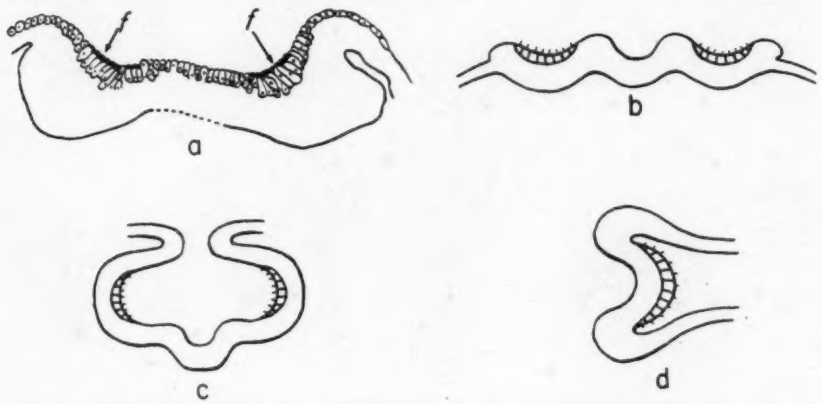

Fig. 6.—The foveolae opticae in relation to Balfour's theory. In *a* is shown the foveolae, *f*, consisting of indented patches of pigmented columnar cells on the open medullary plate of a frog embryo. (From Franz, after Eyclesheimer.) The stages in the evolution of the eyes based on the foveolae are shown in *b*, *c* and *d*. (Redrawn, modified, after Lange.)

the outer wall of the vesicle (fig. 12 *a*), the lateral eyes and the ocular and other evaginations of the diencephalic roof arose subsequent to their involution into the neural tube.

Sleggs (1926) brought forward an interesting corollary. As will be seen later, Balfour's is the only widely accepted theory which accounts satisfactorily for the inversion of the retina, the fact that the rods and cones point away from the light, which must traverse much tissue before reaching them. Far from being an optical detriment as usually supposed, this inversion, Sleggs contended, is most fortunate, for other-

8. I have not been able to see Schimkewitsch's 1910 edition or his 1919 paper in which, as quoted by others, substantially the same views appear to have been expressed.

wise the rich blood supply necessitated by the high trophic requirements of the visual cells would have to be interposed between them and the light. Otherwise the vessels could not get close enough to the outer segments in the visual cell mosaic, for the synaptic and ganglionic portions of the retina must also be adjacent to the rods and cones. Any but very small eyes would thus be impossible unless inversion existed.

Sleggs showed by means of diagrams that the vertebrate eye could have been derived from the integumental eye without the retina becoming inverted (compare fig. 3). He also outlined a derivation of the inverted retina from an erect one, such as is present in the pineal eye, and concluded that the normal inverted condition has a positive significance—it cannot be dismissed as a "defect" of the ocular schema.

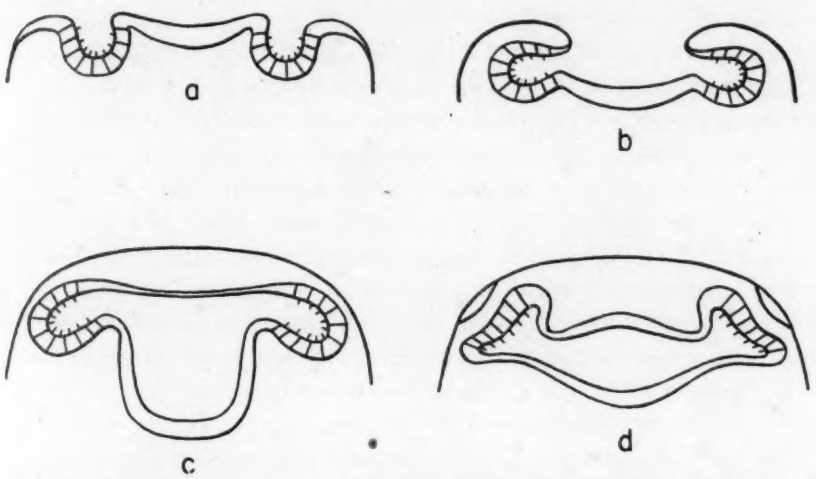

Fig. 7.—Drawings illustrating Schimkewitsch's version of Balfour's theory, deriving the lateral eyes from one of several pairs of photosensory pits in the surface ectoderm. (Redrawn, modified, after Schimkewitsch.)

Estable (1928) also, for other reasons, considered the inversion an actual benefit. It is worth pointing out, too, that if it were not for the inversion the photomechanical adaptive mechanism so prominent in the lower vertebrates could not be made to work so well, for the migratory pigment would then have to be either in the visual cells themselves, as it is in the cephalopods, or, as in the pineal eye, in cells sandwiched between the visual cells and operating to halve the number of the latter per unit area of image. Intracellular color filters (Walls and Judd, 1933) could not have been devised to aid visual acuity. The excavation of the fovea could not have been evolved to spread the image and increase resolving power (Walls, 1937), though of course the prevailing view of this excavation is that it is a necessary evil consequent on the fact of the inversion itself.

Sleggs does not allow himself to become involved in much speculation as to the ancestral conditions of the eye, but he does seem to imply that the retina of the lateral eye was once erect like that of the pineal eye and that it became inverted, so to say, deliberately, the invagination which accomplished this naturally making necessary a new lens. It is unnecessary, however, to suppose that the lateral retina was ever erect simply because the pineal retina is, for it has been abundantly demonstrated by Sleggs himself, as well as by others, that both types of retinas could have come from an ancestral retina which was either erect or inverted. It is interesting to recall that there are even invertebrate eyes, derived from the skin, whose retinas, quite against the rule, are inverted (Patten, 1886). Most of the objections to Balfour's theory have been directed at the foveolae opticae. By many, for example, Tretjakoff (1913) and Studnička[9] (1918), these are considered precocious and cenogenetic formations. The eyes exhibit this early sign of their development simply because they have so far to go to reach their complex maturity. The foveolae are not vestiges of the phylogenetic past but rudiments of the ontogenetic future, with no more significance for evolution than the precocity of development of the central nervous system itself.

Studnička objected that the columnar cells which line the foveolae (fig. 6 *a, f*) do not even remotely resemble the rods and cones, hence can have no photosensory function, present or past. This is specious, however, for the rods and cones do not even appear at all when one might (with phylogeny in mind) expect them to, but much later. They are the last cells in the whole retina to differentiate, for the layer from which they form has first the task of proliferating the remainder of the retina, and the rods and cones cannot be matured until mitosis has ceased. Yet historically, though not ontogenetically, there must have been visual cells before there was an optic cup. The foveolae could not reasonably be expected to resemble the adult retina, even if their significance is all that Froriep, Lange and others have thought it. It needs pointing out, however, that the foveolae may be entirely ignored without at all affecting the plausibility of Balfour's original concept.

Studnička's other principal objection carries rather more weight. In all of the fishes except the elasmobranchs, the central nervous system originates as a solid structure which gains its lumen secondarily. There are no foveolae opticae in such species, and considering the likewise solid anlages of all invertebrate nervous systems, the piscine method of neural tube formation may well be the more primitive one. If so, Balfour's theory is eliminated as an explanation of the inversion of the retina, for the inversion is not then due to inheritance from an involuted integu-

9. Studnička (1918) called attention to the fact that a strikingly similar situation occurs in cephalopod molluscs, where of course it can have no meaning for vertebrate evolution.

mentary source. Parker's (1908 *b*) objection on other grounds will be treated later in the consideration of Boveri's theory. Incidentally, Boveri's and Studnička's theories (v. i.) are both equally workable, whether one believes in a solid or a hollow primary condition of the chordate nervous system.

The supposed deep significance of the foveolae opticae has had its champions even recently. Franz (1934 *a*) expressed the belief that they might be set down as mere precocities if it were not for the pigmentation of their cells in certain animals. This plays no part in the subsequent

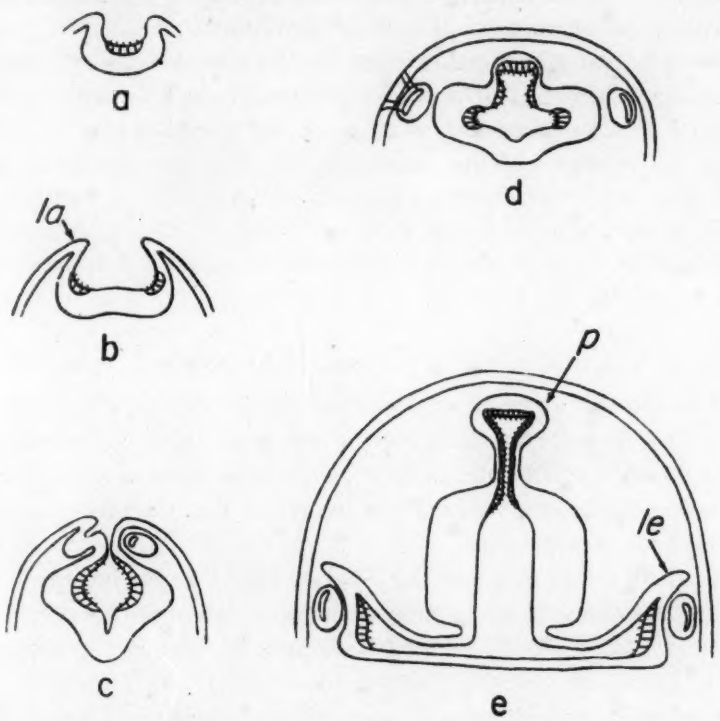

Fig. 8.—Franz's schema of the evolution of the pineal and lateral eyes. The Amphioxus infundibular organ stage is shown in *a;* the foveolae opticae stage, showing the future lentogenic area, *la,* in *b* and the stage of general diencephalic photosensitivity in *c*. The lens is shown in an earlier condition on the left and a later condition on the right; this also applies in *d,* in which the lentogenic areas have shifted laterad and the epiphysis is distinct from the lateral ocular evaginations. In *e* is shown the final situation with the pineal eye, *p,* and the lateral eyes, *le,* differentiated, and with residual photosensory tissue in the stalk of the epiphysis and the neighboring diencephalic roof. (Redrawn, modified, after Franz.)

formation of the definitive retina. . He considered it to be reminiscent of an actual photosensory function of similar cells, similarly located, in the past; for the visual cells of many tunicates are likewise pigmented. Franz offered diagrams (fig. 8) which show a derivation of the lateral

eyes from a median photosensory ectodermal pit in the floor of the invaginated but unclosed neural tube. He derived the lentogenic area from the junction of the margin of the optic pit with the surface ectoderm and in this way (personal communication) accounted for the regeneration of the lens from the dorsal rim of the secondary optic vesicle. According to this view, the lens is closely related to the neural crest. In this connection, the homology claimed for optic vesicles and neural crests by Conel (1938) becomes interesting, and, in turn, the evidence of DuShane (1935) for a neural crest origin of all pigment cells is perhaps pertinent. Even the capacity of lens tissue to form melanin in cataract may have a modicum of phylogenetic significance!

The only really vulnerable point in the present theory is its need of assuming continuous photoreception throughout the transition from a superficial to an endoneural location of the visual organ, in order to explain the inversion of the retina (fig. 5). It has already been mentioned that two other theories account as well for the inversion, and as shall be seen later from the nature of rods and cones, their ancestors as far back in time as the surface ectoderm could not have been continuously sensory.

VON KUPFFER'S THEORY—THE EPIBRANCHIAL PLACODES

While Balfour's theory accounts for the cerebral origin of the retina and for its inverted condition, leaving the lens out of consideration, the placode theory explains the history of the lens explicitly and plausibly; but it is so unsatisfactory as regards the retina that the theory as a whole has long been in disrepute.

This theory was proposed by Sharp (1885) as an interpretation of the lens. Its value in connection with the problem of the origin of the retina was independently seen by several investigators, including Nuel (1887), Béraneck (1890), Burckhardt (1901) and Lubosch (1909). Credit for the theory is usually given to von Kupffer (1894). Briefly, it is as follows:

The ancestor of the vertebrates bore in the region of the head a series of paired sense organs comparable to, and probably homologous and serial with, the lateral line organs. From anterior members of this series, which are seen embryologically as ectodermal placodes, the olfactory organ, the membranous labyrinth of the ear and the lens of the present lateral eye were derived. The lens was formerly a vesicular eye in its own right, with an erect retina, of which the present retina was the ganglion (fig. 9). This ganglion took over the sensory function phylogenetically and released the older eye to serve as its definitive dioptric accessory. The cup form of the definitive retina is thus accounted for, and the new complex, being capable of eikonogenesis, was much superior to the original purely directional eye.

The difficulties involved in the conversion of the hypothetic ganglion into the present retina are insuperable, as many have pointed out. The placode theory cannot account even for the inversion of the retina (Parker, 1908 *b*), to say nothing of its structure. There is no embryologic evidence for a previous connection of the present retina and lens,[10] although Lubosch (1909) was misled by the aberrant arrangement of the inner layers of the retina of the lamprey into thinking that such evidence exists. The only tissue which ever lies between the optic cup and the site of the lens placode is mesodermal, and it is only where there is no such tissue that the lentogenic thickening occurs. Franz (1934 *a*) entertained Hescheler and Boveri's (1923) "histologic" evidence for a former visual function of the lens, consisting of an occasional scattered

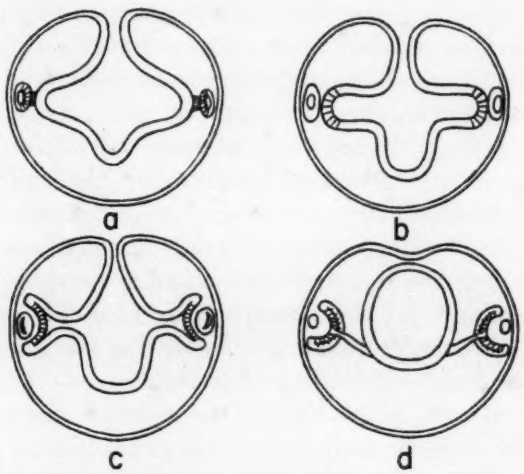

Fig. 9.—Drawings illustrating the placode theory. A vesicular eye derived from a lateral line organ loses its photosensitivity and becomes the definitive lens, while its ganglion becomes photosensory and is converted into the definitive retina. (Redrawn, modified, from Keibel, after Béraneck.)

pigmentation together with the rodlike form of the vitreal ends of the lens fibers; but this is no evidence. The lens may once have been an organ, even a sense organ, but there is not the slightest actual evidence that it was ever an eye.

10. Tretjakoff's (1913) belief in such a connection (fig. 4 *d*) was held for reasons which bear no relation to the placode theory, and the connection he visualized was imagined to have been made secondarily. The delicate bridges which temporarily connect the optic cup with the lens vesicle and which contribute to the primary vitreous are, as Miss Mann (1928) pointed out, matched by similar protoplasmic strands between cell layers throughout the embryo and have no particular meaning for the eye as such.

Some of the more ardent proponents of the placode theory have been at some pains to homologize the retina with a spinal ganglion.[11] Thus Nuel (1887), who compared the entire eye with a lateral line organ,[12] has claimed this homology and likened the optic nerve to the dorsal root of a spinal nerve despite its apparent ventral origin.

Although the idea of an intimate past relation between a sensory predecessor of the lens and a nonsensory forerunner of the retina has been generally abandoned, the convenient interpretation of the lens as a formerly independent structure has persisted and has been repeatedly used by those who cannot otherwise account for the lens in their own hypotheses. Jelgersma (1906) and Studnička (1918) have done so,[13] and Schimkewitsch (1921), particularly, found this phase of the theory indispensable. Holding the dorsal position of the solid teleostean primary optic anlages to be the primitive position and pointing to the (dorsal) median eyes and to von Kupffer's comparison of the "dorsal cells" of Amphioxus (fig. 1, *dc*; these are now known to be photosensory, v. i.) with an embryonic neural crest, Schimkewitsch compared the embryonic optic vesicles with spinal ganglion anlages and derived the definitive ("outer") lens from and anterior placode of the epibranchial series. He considered this association reminiscent of the relation of the ganglion anlages of the head with placodes. It has already been seen that this author derives the retinas of the lateral and median eyes from metameric ectodermal pits (fig. 7). He thus gives a picture of an involuted lateral line organ (the eventual retina) commandeering still another lateral line organ (the eventual lens) to serve as its dioptric body. The muddy waters are not cleared when one asks from which "metamere" the lens may have been derived, considering that according to Schimkewitsch

11. See Jelgersma (1906), who likens the rods and cones to the neuroepithelial portion, and the retinal bipolar layer to the spinal ganglions, of other sense organs. He considered that the retina as a whole cannot be compared with other sense organs since it includes the entire peripheral and central nervous portions of other sensory complexes. It is nowadays more the fashion to compare the layers of the retina with those of the brain wall (Mann, 1928); but with the exception of the single (cellular) layer which they share in common, the ependyma, the lamination of the retina is unique and is expressive only of its own physiology.

12. He was perhaps misled in his choice by the mistaken supposition that the photogenic organs of the lateral line in deep-sea fishes are eyes, which indeed they can strikingly resemble in structure.

13. However, they have done so with these differences. In Jelgersma's thesis the skin lens is obtained by the retina before its own ("retinal") lens ceases to function; whereas according to Schimkewitsch, it is because the "retinal" lens has been rendered useless by the invagination of the retinal vesicle that the "outer" lens becomes necessary (fig. 12). In Studnička's hypothesis no inner, retinal lens is involved. The vertebrate eye has had two lenses in its history (fig. 3 *a, b', c' and d'*) according to Froriep (1906 *a*), but he does not suggest a source of the second, definitive lens.

the lateral retinas stem from the foremost pair of ectodermal pits, and those pairs just behind them develop eventually into the retinas of the ocular evaginations of the diencephalic roof.

The actuality of the embryonic lens placode itself has been challenged. Is there really any such thing as a morphologic entity? If, as has been known for years, a lens will not develop if the optic cup is removed or prevented from contact with the skin, and if a lens will form from almost any ectoderm under which an excised optic cup has been placed, need one then believe the formation of the normal lens anlage to be anything more than an immediate chemotactic response—of no more phylogenetic import than a blister?

On the foregoing experimental basis, Keibel (1906) considered the lens to be an entirely secondary structure, called into existence phylogenetically, as it is ontogenetically, by the evolving optic cup. Just how, he did not suggest. Tretjakoff (1913), who is likewise unconvinced of a lens placode, offered a mechanical explanation: The evolving retinal cup became affixed to the ectoderm and at the junction muscle cells developed (fig. 4 *d, m*), which made possible a primitive sort of accommodation. The pull of these muscle cells (which later developed into the accommodatory retractor lentis muscle of the fishes, the campanula Halleri) evoked a lentoid thickening—a sort of callus—of the ectoderm (fig. 4 *d, l*) and its eventual separation as a free lens (fig. 4 *e*). This seems plausible until one realizes that there would be no point to accommodation in an eye in which there was no image to be focused on the retina. The muscle which Tretjakoff considered produced the lens would only have arisen after a respectable lens had already been evolved. Moreover, the campanula is lacking in the lowest vertebrates, the lampreys, whose accommodation is brought about entirely by extraocular muscles (Franz, 1934 *a*). It is most improbable that any intraocular muscles antedated the lens in evolution.

The valuable review of Mangold (1931) summarizes instances in lower vertebrates in which a lens has partially or completely developed when the retinal anlage of the cephalic plate had previously been removed. He referred also to the occasional occurrence of lenses in anophthalmic terata. Mangold expressed the belief that the field of experimental embryology could offer support to von Kupffer's theory only if a potential interchangeability of the lens anlage with the other, veritable, head placodes could be demonstrated. If a self-determined ectoderm-thickening stage occurred, subsequent to which the special determinations took place, one could conclude that the formation of the lens independent of the optic cup is a more primitive process than the usual, dependent, development. Such experiments in interchange of placodes as have so far been performed, however, speak against an intrinsic

determination of the lens anlage. Mangold concluded that the self-differentiating power of the lens is so low that the open question must remain open. Since his review there have been a few additional reports of a lentoid thickening of the ectoderm or of somewhat more of a lens being formed in the absence of the optic cup (Choi, 1932; Waddington and Cohen, 1936).

A negative phylogenetic import of the apparent immediate dependence of the lens anlage on the optic cup can therefore be neither denied nor completely affirmed. All considered, the placode theory still offers an explanation of the lens—grounded so far in the past that concrete evidence against it can hardly be hoped for by its severest critics, however little appeal it may have for them.

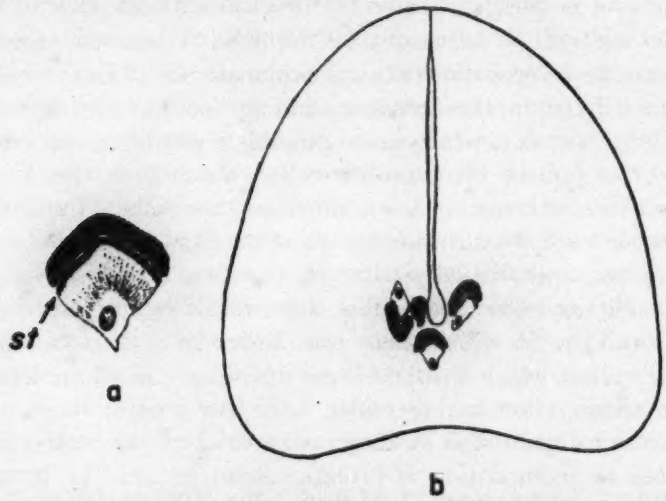

Fig. 10.—The Hesse organs of Amphioxus. In *a* is illustrated a single organ showing the pigment cell and the photosensory ganglion cell with its *Stiftchensaum, st.* In *b* is shown the orientation of several organs about the central canal in a transverse section of the nerve cord. (Redrawn, modified, after Hesse.)

BOVERI'S THEORY—THE PHOTORECEPTORS OF AMPHIOXUS

The theory of Boveri was the first to point to an actual, existing type of photosensory cell as the ancestor of the vertebrate rods and cones. Like the ascidian hypothesis, this theory postulates that the vertebrate retina originated endoneurally and is thus at first thought irreconcilable with Balfour's theory.[14]

14. However, Jelgersma expressed the belief that it is unreasonable for a sense organ to originate so distant from its stimulus and that Hesse's organs must originally have been derived from the skin. It is difficult to see, however, how a photoreceptor can well be "distant from its stimulus" in a transparent animal!

In 1898 Hesse investigated the tiny pigmented organs which are scattered along the posterior portion of the nerve cord of Amphioxus and found each of these to consist of a pyramidal ganglion cell embraced by a pigment cell which cuts off the light except from certain directions. Variously oriented, these directional eyes lay a basis for the strong negative phototropism exhibited by the animal (fig. 10). If proof, beyond logic, of the photosensory character of "Hesse's organs" is required, it is furnished by the experiments of Parker (1908*a*) and Crozier (1917). The distribution, orientation and cytologic structure of these organs have recently been studied by Franz (1923), Joseph (1928), Kolmer (1928) and Wollenhaupt (1934).

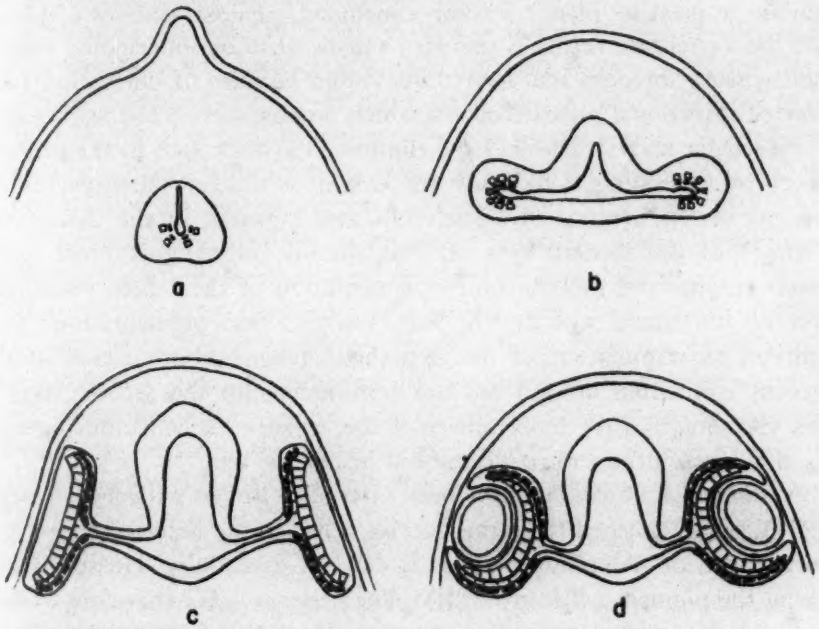

Fig. 11.—Drawings illustrating Boveri's theory. The Hesse organs of an Amphioxus-like ancestor became carried out into two lateral evaginations, which then invaginated. In the outer layer of the cup thus formed the ganglion cells disappeared, while in the inner layer they evolved into the rods and cones, the pigment cells disappearing. (Redrawn, modified, after Boveri.)

T. Boveri (1904) made Hesse's organs the basis of a theory of ocular origin which has proved quite popular (fig. 11). Boveri supposed that in an Amphioxus-like ancestor the two-celled visual organs became oriented with their pigment cells all toward the central canal, and congregated on the ventral side of the anterior end of the neural tube. From this at first median anlage, paired lateral diverticula carried the visual organs toward the skin. In the distal walls of the diverticula the pigment cells lost importance and disappeared, whereas in

the proximal walls they remained to form the retinal pigment epithelium while the sensory ganglion cells vanished. Invagination now completed the cupuliform vertebrate retina, the inverted structure of which is well explained.

Boveri's theory has been supported by Parker (1908 *b* and 1909) and by Hescheler and V. Boveri (1923) and is favored by Tretjakoff (1913) and Nowikoff (1932). Parker emphasized that the sensory cell of the Hesse organ is inverted with respect to its pigment cell and considered the variable attitude of the organ an indication that all orientation has been lost since the ultimate derivation from the skin. It might be objected, however, that although the position of a given organ seems haphazard, the orientation of the entire group of organs exhibits a positive plan. Parker concluded, against Balfour's theory, that the vertebrate retina is inverted not because of inheritance from a photosensory integumental source but rather because of derivation from inverted directional photoreceptors which became alined and aggregated.

Hescheler and V. Boveri have eliminated a weak spot in the original theory by extending it to cover the origin of the median eyes. They stressed the occurrence of visual cells and pigment in the distal wall ("lens") of the median eyes of Petromyzon and Anguis (just as T. Boveri emphasized the transitory pigmentation of the future visual cell layer of the lateral eye in frog embryos). These pigmentations they regarded as reminiscent of the hypothetic former continuation of the pigment epithelium around the ora terminalis into the sensory retina. This view might have more appeal if the sensory element and pigment cap of a Hesse organ were all one cell instead of two.

A number of objections have been offered to Boveri's theory. Joseph (1904), who identified the large "dorsal cells" in the anterior end of the neural tube of Amphioxus (fig. 1, *dc*) as essentially Hesse organs lacking the pigment cells, favored Müller's theory. He therefore looked for no phylogenetic significance in either Hesse's organs or the photosensory [15] "Joseph's cells," which he himself elucidated. Joseph pointed out that there are no Hesse organs whatever in the region of the brain and that where visual organs are excluded from the region of the head in other animals this is always a secondary phenomenon. Naturally, he also questioned the propriety of claiming such precise homologies between the vertebrates and such an offshoot as Amphioxus.

Jelgersma (1906) objected that no ontogenetic stages confirm the theory, and Keibel (1906) criticized it on similar grounds, stressing the lack of intermediate stages among adult chordates. Since the Hesse organs are lacking in the anterior segments, said Keibel, one

15. Wollenhaupt, 1934.

should have to believe that the region of the head of Amphioxus is degenerate if one is to support Boveri's theory. This necessity is transformed into a virtue by Hescheler and V. Boveri, who accounted for the absence of anterior Hesse organs by assuming just such a rudimentation of the brain. These authors considered that the dwindling of the chordate brain left the eyes behind, so to say, against the skin; thus the ontogenetic evagination of the optic vesicles is without evolutionary meaning.[16] In extension of this view they assumed a dermal armature of the head of the provertebrate, which would render a historical "evagination to keep near the stimulus," *à la* Balfour, ridiculously useless. Needless to say, however, there is not an iota of paleontologic evidence for their cephalic scutation.

Schimkewitsch (1921), who favored a dorsal origin of vertebrate eyes, is naturally unsympathetic toward Boveri's theory, since the latter postulated a ventral origin. It could have been as easily devised to fit the idea of dorsal origin, however. Schimkewitsch further claimed that the theory cannot account for the invagination of the lateral eyes and the failure of the median eyes ever to invaginate. His own (1902 and 1921) explanations of these points are, however, neither convincing nor incapable of reconciliation with Boveri's hypothesis.[17]

Despite criticisms, the theory is ingenious and plausible. It accounts nicely for the inversion of the retina and offers a tangible prototype of the rods and cones. Therein lies its supposed great strength and its actual great weakness. Some have been able to see actual cytologic resemblances between the Hesse organs and rods and cones; but the principal imagined point of resemblance, the *Stiftchensaum* (fig. 10*a*, *st*),

16. Compare the similar view of Tretjakoff (1913), based on the early ideas of His.

17. Schimkewitsch supposed that as the lateral eyes swung outward and ventrad into their definitive position they were passively indented ventrally, thus forming the embryonic fissure, by the resistance of tissue in their pathway (fig. 12). The lateral eyes were thus carried laterally from their original dorsal and paramedian position by the same process which has caused the cranial nerves to take up a ventral departure, i. e., the growth of the dorsal brain wall. He expressed the belief that the migration of the eyes has ontogenetic reflection in the invasion of the embryonic fissure by mesoderm which plays no subsequent part in the elaboration of the ocular tissues *sensu strictu* and explained the optic chiasma by suggesting that the ventrad duction of the optic nerves was so strong that the process "overshot." Tretjakoff (1913) quoted and supported Vialleton's criticism in this connection, asking why only one pair of eyes became displaced laterally, and decried the brutal "passive invagination" as quite foreign to the delicacy of most developmental movements. Schimkewitsch, however, made the point that the roof plate of the diencephalon has never expanded laterally; hence the pineal and parapineal eyes have remained in their original location.

has been claimed to be an artefact by Franz (1923) and Wollenhaupt (1934)—see, however, Joseph (1928) and Kolmer (1928). Whether it is an artefact or not, there is no close comparability of the two types of cells. While this in itself is not fatal to Boveri's theory, it will be seen later that Amphioxus possesses photosensory cells which are far more closely comparable with vertebrate rods and cones than are its Hesse organs or its Joseph cells.

STUDNIČKA'S THEORY—THE PHOTOSENSORY EPENDYMA

As early as 1875 Krause suggested a homology of the rods and cones with the ependymal cells of the brain lining. He conceived of a fusion of the embryonic ependymal cilia into bundles, each of which became the outer segment of a visual cell. Such a compound origin of the rods and cones was finally made untenable by the precise histogenetic research of the decade of 1904-1914. Even before then, the notion had

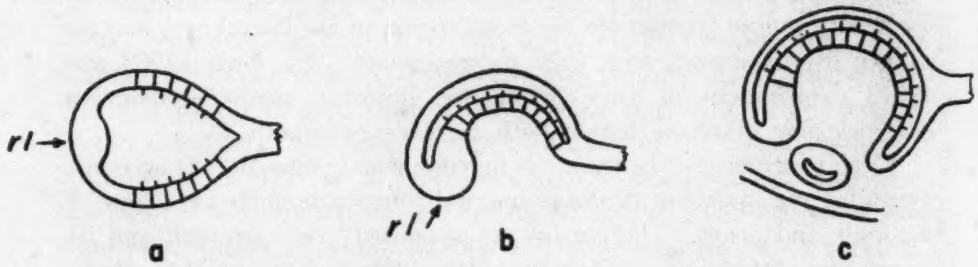

Fig. 12.—Drawings illustrating Schimkewitsch's explanation of the origin of the invaginated optic cup, the embryonic fissure and the need for a second lens. The original retinal lens, *rl*, of the ancestral vesicular eye (*a*) is uselessly located after the eye has been indented (*b*) by its ventrad migration through resistant tissue, so that a new lens becomes necessary (*c*). (Redrawn, modified, after Schimkewitsch.)

little appeal and was rejected by Studnička (1900) in his monograph on the ependyma. At that time Studnička homologized the retinal Müller fibers with ependymal and neuroglial cells but said of the rods and cones that they "apparently had no counterparts in the structure of the central nervous system."

Studnička later, however (1912, 1913 and 1918), advanced and elaborated an ocular phylogenetic theory based on a derivation of the visual cells from ependymal cells. This theory has been unable to bear the weight of Krause's original misconception (despite Krause's prompt abandonment of it in 1876), with which it is persistently confused to this day. There is no current textbook of any description in which the rods and cones are termed modified ependymal cells; they are everywhere called "neuroepithelium" or "neurons." The most recent monographers of the retina either ignore the ependymal theory entirely or state that it

"does not appeal" (Arey, 1932 *a* and 1932 *b*). It shall be seen that when all the evidence is assembled the appeal is irresistible; but the reason for the present paradoxically poor position of the theory is clear. Its sole modern proponent (Studnička) simply failed to adduce more than a fraction of the favorable evidence which lay to his hand.

Studnička compared the rod and cone layer with the ependymal layer of the brain wall by analogy of position. He pointed to a number of sense organs of the central nervous system containing sensory ependyma, by way of precedent for the rods and cones being similarly interpretable. He accepted the placode theory for the origin of the lens and laid great

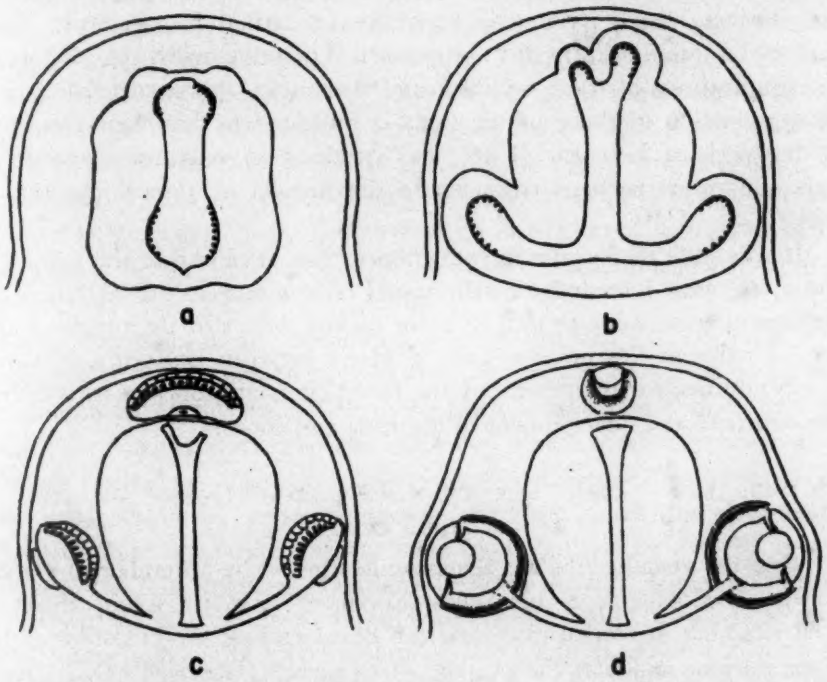

Fig. 13.—Drawings illustrating Studnička's theory of the derivation of the pineal and lateral retinas from the ependyma. In *c* is shown the young lamprey larva in which the retina is apparently functional though the optic vesicle is still "primary" and the lens is flat and functionless. (Redrawn after Studnička.)

stress on the condition of the precociously functional eye of the young larval lamprey as indicating that the vertebrate eye passed through a period as a directional eye before it became capable of image formation. The flatness of the lens of this functional proammocoete eye—its incapability of dioptric function—led Studnička to believe that the lens must have been present in evolution as a functionless vestige of something else before the photosensitive portion of the brain wall ever evaginated to become the retina. His whole theory embraced the median as well as the lateral eyes (fig. 13), for, as he showed, the sensory cells alternate

with nonsensory ependymal cells in the retinas of the median eyes of the lamprey.

Without more evidence than Studnička himself presented, one would be unable to choose between his explanation of the retina and Boveri's; indeed, Studnička himself thought that the Hesse organs and the Joseph cells of Amphioxus might have had ependymal origin. Only Parker (1909) and Tretjakoff (1913) have insisted on an endoneural origin of vertebrate photoreceptors, and even the latter inclined toward Boveri's theory rather than toward Studnička's, which has been announced since Parker's publication.

The body of evidence which Studnička might have employed, but did not, derives largely from the literature of retinal histogenesis. The authors of this literature did not concern themselves with the phylogenetic implications of their findings, and Studnička's own surprising lack of appreciation of those implications is indicated by his brief reference to the work of Leboucq (1909) as "speaking at least for considering rods and cones perhaps traceable to the flagella of typical ependymal cells."

It was indicated in the introduction to this review that the cytologic and cytogenetic knowledge of the visual cells would, in the last analysis, demonstrate the value or lack of value of any theory of the origin of the eye. Unable at this point to choose wisely between Balfour's, Boveri's and Studnička's explanations of the retina, it is appropriate to consider the structure and development of the rods and cones.

INDICATIONS FROM THE CYTOLOGY AND CYTOGENY OF THE VISUAL CELL

That the visual cell is not a neuron is attested by a number of points. In its development, a neuroblast produces first its axon, then its dendrites; but an immature visual cell develops first the cytosome distal to the nucleus and later the axonoid "foot piece" (Greeff, 1900). Visual cells develop in the germinative layer of the retina. Neuroblasts never lie in the corresponding layer of the brain wall. Adult visual cells possess centrosomes, which neurons never contain (Penfield, 1928; Wislocki, 1928). No neurologist has ever identified neurofibrillae in visual cells (Embden, 1901; Vogt, 1902; Ramon y Cajal, 1904; Bielschowsky and Pollack, 1904; Vermes, 1904; Rozemeyer and Stolte, 1930).

The rods and cones have also been called "neuroepithelium" since the time of Schwalbe (1874), who first thus designated them. This term is in line with Balfour's theory but would be deserved only if it could be shown that their ancestors as far back as the dorsal ectoderm location were continuously sensory. Since every cell in the body is, of course,

to the same extent traceable back to epithelium, the "sensory" aspect needs stressing.

To think, instead, of the visual cells as sensory ependyma need occasion no shock to the imagination. Ependymal cells may have synapsoid connections with true nerve cells (Pensa, 1904). Studnička, in one of his papers, gave an exhaustive list of receptor organs of the central nervous system the sensory elements of which are ependymal cells.[18] Ordinarily, these are in direct relation to ganglion cells. In the lateral retina, bipolar and amacrine cells have simply been interpolated to serve summative, associative and inhibition-excitation functions.[19] Not only are there many cases of sensory ependyma, but some of photosensory, even if the pineal retina is not considered. Thus on experimental grounds, von Frisch (1911), Scharrer (1928), Nowikoff (1934), Young (1935) and Benoit (1937) are convinced of the photosensitivity of certain parts of the brain, notably the diencephalon; and some of these investigators have localized the reception in the ependyma. In Amphioxus there is photosensory ependyma—that of the median "infundibular organ" (fig. 1, *inf*)—which is shadowed differentially by the anterior pigment spot as the animal or the light source moves (Franz, 1912 and 1923).

The functions of the ependyma at large are support, secretion and circulation.[20] It might be argued that before the rods and cones are designated as ependyma, other more likely candidates should first be sought among derivatives of the optic cup which serve one or another of these functions. The pars caeca retinae is secretory, but there are no cilia or flagella in the eye for circulatory purposes, convection alone being adequate, supplemented in many eyes by surging produced by movements of the lens in accommodation. Indeed, the intraocular portion of the cerebrospinal canal is obliterated, and in the adult there is little fluid in its homologue (the interbacillary spaces) and no need for it to be circulated.

The pars optica retinae does, however, possess sustentative elements, some of them undoubtedly glial. The most conspicuous are the Müller

18. However, Agduhr (1932) inclined to the view that in all such organs there must be ectopic neurons which are the actual receptors.

19. Tretjakoff (1913) considered that the absence of such elements from the pineal eye destroyed any comparability of the latter with the lateral eyes; but if the identical origins of the respective sensory elements are kept in mind, it must at least be admitted that the two types of eyes are homologous if not actually serially homologous.

20. It has been vigorously denied that ependymal cilia and flagella are motile (Fuchs, 1902, who considered them hygrophoric stereocilia adaptive to the secretory activity of the cells), but Stoklasa (1930) and his witnesses have watched them beating in many animals.

fibers, which are usually considered neuroglia and have even been assigned to the category of protoplasmic astrocytes by Marchesani (1926). But as was already known to Babuchin (1863), they are the first elements of the retina to differentiate. They maintain connections with both limiting membranes (which indeed they generate) throughout life. These are characteristics of the ependymal cells, which form so early and act as rivets in the wall of the young neural tube (Studnička, 1900 and 1912). The Müller fibers therefore best deserve to be considered specialized ependymal cells; but such designation does not preclude a similar interpretation of the visual cells.

Accurate knowledge of the cytogenesis of the rods and cones began with the work of Fürst on the salmon in 1904. This investigator found that the simple epithelium of the optic cup became pseudostratified by the proliferation of a number of cell generations from the original, germinal layer. Each cell for a time retained connections with both limiting membranes, but in all except the Müller fibers and the visual cells both of these connections were eventually broken or retracted and the centrosomes discarded, the cells differentiating into the various neural elements of the retina. The visual cells differentiated from the cells of the germinal layer after the cessation of mitosis. Thus the visual cell layer is the germinative one of the retina just as the ependymal layer generates the brain wall.

Fürst on the salmon, Leboucq (1909) on six species of mammals, Magitot (1910), Seefelder (1910) and Mawas (1910) all independently on the human being and Leplat (1913) on the chick all describe the cytogeny of the visual cell in essentially identical terms. The early embryonic coating of fine stereocilia, which in some forms is present over the sclerad surface of the inner layer of the optic cup, is apparently soon lost,[21] and each epithelioid young visual cell, when it commences to differentiate, is nude and contains a double centrosome lying toward the cleft between the retina proper and the future pigment epithelium. From and of this diplosome a naked filament develops, to protrude from the cell as the anlage of the outer segment (fig. 14 *a*). As this thread lengthens, the cytoplasm of the cell bulges at its free surface to form the anlage of the inner segment. From this peninsula, mitochondria migrate out and encrust the centrosomic filament (Fürst's fiber),

21. It was a clumping of these structures which Krause (1875) at first imagined to form the rod and cone cytosomes, and the present neglect of the ependymal theory is largely due to the widespread knowledge of their transitory character. They are not true cilia and are not provided with basal granules, since during the time they are present the centrosomes are occupied with mitosis. They are gone by the 10 mm. stage in the human embryo (Mann, 1928), and it is not until the 34 mm. stage that the centrosomic filaments which are the visual cell outer segment anlages appear (Seefelder, 1910).

depositing in a spiral organization which is retained by their descendant material into the adult condition (Franz, 1934 *b*). The diplosome remains always just within the outer segment, and from it a second, short fiber (Kolmer-Held fiber) eventually grows part way down through the inner segment like a vestigial tonofibril.

In its development, the visual cell thus behaves remarkably like any flagellated cell; indeed, Leboucq, Magitot and Leplat all remarked on the similarity of develpoment of an outer segment of a visual cell and a sperm tail (fig. 14 *a* and *b*), though none of these investigators gave any sign of a phylogenetic consciousness. The adult outer segment is composed of an axial centrosomic filament surrounded by a thick lipoid envelop of largely mitochondrial origin.[22]

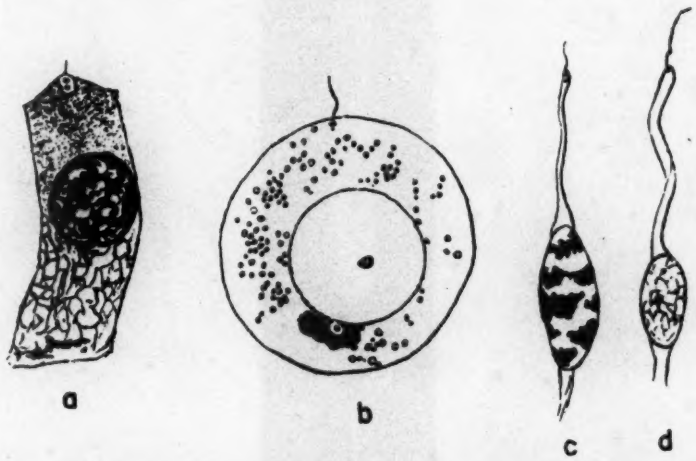

Fig. 14.—Comparison of young visual cells with flagellated and ependymal cells. In *a* is depicted a foveal cone of a 34 cm. human fetus, showing filamentous anlage of outer segment proceeding from diplosome (redrawn after Seefelder); in *b*, a young human spermatid showing anlage of flagellum consisting of diplosome and centrosomic filament (redrawn after Gatenby and Beams); in *c*, the immature fundal cone of a kitten (redrawn after Lebouck), and in *d*, the ependymal cell of a carp (redrawn after Franz).

The descriptions of the histogenesis of the retina and of the brain wall are identical; the respective germinative roles of the visual cell layer and the ependyma are the same, and the visual cell differentiates as a flagellated cell (fig. 14 *c* and *d*), the outer segment being the homologue of the flagellum and the intersegmental junction representing the ancestral free border of the cell (fig. 15). The immediate ancestors

22. It would be unfair, however, to lay emphasis on the spiral organization of the mitochondria, for such organization is no longer widely credited to the mitochondria of the massive flagellum of the spermatozoon.

of the rods and cones could only have been flagellated cells, and the only such cells which could possibly have become the visual cells were those of the provertebrate ependyma.

THE ORIGIN OF THE RETINA

This acceptance of the ependymal origin of the visual cells necessitates the complete rejection of Boveri's theory, for Hesse's organs and Joseph's cells are sensory ganglion cells; but it remains to be decided whether Balfour's and Studnička's hypotheses can be combined.

Fig. 15.—Generalized vertebrate rod visual cell, showing from above downward: cylindric outer segment containing a myeloidal spiral and centrosomic Fürst fiber; intersegmental junction; inner segment containing refractive ellipsoid, diplosome and Kolmer-Held fiber connected therewith; external limiting membrane; a portion of rod foot piece containing nucleus. (Original drawing.)

The ependymal cell is an ancient component of the chordate central nervous system. Although in most vertebrates the thickness of the brain wall is such that neuroglial elements are greatly predominant in sustentation, the ependymal cells in cyclostomes are able to bridge the whole thickness and there afford the principal support. In Amphioxus they are the sole connective elements of the neural tube (Agduhr, 1932).

Yet there must have been a neural tube before there was ependyma. The ependymal concept of the rods and cones brings them into line with sensory hair cells in general, and it is important to note that the essential

organelle is the outer segment—the "hair." Before there was flagellated ependyma there was no endoneural anlage of the vertebrate retina, and photosensory ancestors of the flagellated ependymal cell are scarcely to be sought. Ependyma, flagellated or not, was first made necessary by the fluid-filled tubular nerve cord which, as has been seen, may not even have been primarily tubular in chordate evolution. The ependymal cell does trace back to the dorsal ectoderm historically, and there were

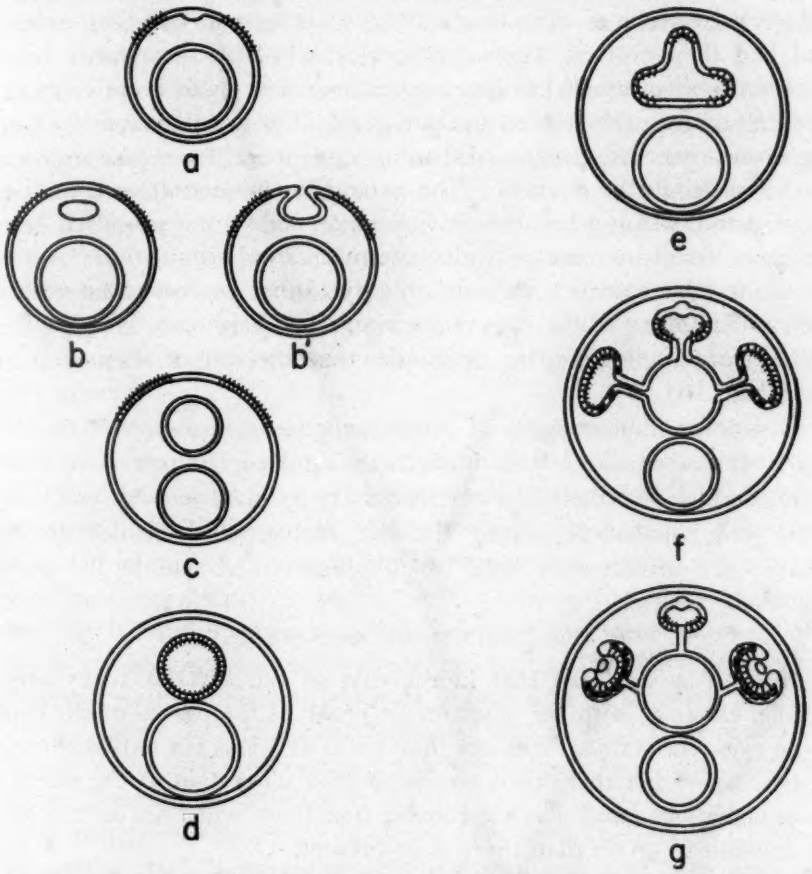

Fig. 16.—Drawings illustrating the author's conception of the evolution of the pineal and lateral eyes. The prochordate stage with photosensory ectoderm and the nerve cord not yet separated from the skin is shown in *a*. The alternative stages in evolution of the neural tube, depending on whether one adheres to the "solid" or "hollow" theory, are shown in *b* and *b'*. In *c* is shown the tubular nervous system formed but with the ependyma purely sustentative, secretory and circulatory. In *d* the ependyma has locally become photosensory and the photosensory cells have disappeared from the skin (provertebrate stage). The drawings *e*, *f* and *g* are self explanatory and show dorsal and lateral patches of photosensory ependyma evolving into the pineal and lateral retinas. (Original drawings.)

likely photosensory cutaneous cells which disappeared on the advent of the superior definitive visual organs without being concerned in their formation. But since the ependymal cell was phylogenetically differentiated primarily for support, secondarily for secretion and tertiarily for circulation, it is impossible to believe it to have been sensory from the first (fig. 16).

The fact that even the limicolous and photophobic Amphioxus requires no dermal sensitivity is reason enough to conclude that the pelagic provertebrate, once its nonsensory neural tube had been produced and had then evolved flagellated ependyma which secondarily became photosensory, proceeded to lose any rudimentary photoreceptive capacity its skin had formerly needed and possessed. The great advantage, operating as an urgent reason, for substituting endoneural for ectoneural photoreception should be obvious. The protected endoneural receptor could proceed to develop a high sensitivity, since, unlike the primitive dermal receptor, it was immune to inadequate mechanical stimulation [23] with its attendant false alarms. Balfour's theory cannot be reconciled with the ependymal nature of the rods and cones. The vertebrate retina did not evolve from anything earlier or simpler than the wall of the neural tube itself (fig. 16).

The infundibular organ of Amphioxus, as Krause (1876, p. 750) clearly suspected, is an indubitable homologue of the paired vertebrate retinas, which got their phylogenetic start as photosensory patches of brain wall somewhere among the lost forms which bridge the gap between the evolutionary level of Amphioxus and that of the ancient cyclostome fishes.

THE ORIGIN OF THE LENS

It is to be deplored that in the case of the lens no such ease of decision exists as with the other major phase of the puzzle of the origin of the eye—the retina. It is not that one is at a loss for explanations of the lens but rather that one is unable to be at all certain of the worth of the explanations; and it is improbable that there will ever be any more information to go on than there is at present.

It has been shown that by far the most of the theorists have appealed to the placode theory for an account of the lens. The retrospect of a lateral line organ, becoming functionless but lingering on in the body until captured by the evolving retina, is a tantalizing one. It is almost a pity that the tangible evidence for it—the occasional slight self-differentiatory capacity of a lens anlage—is so weak. It has seemed to some an important question whether the organ formerly constituted by the

23. By this is meant the evocation of a sensation by a stimulus which the receptor is not specifically designed to receive, the experience being interpreted as due to the other, more usual kind of stimulus.

lens had become functionless or not when the optic cup took it over. The one view was held by the proponents of the placode theory in its entire classic form. The other point of view is represented by those who hold other opinions as to the history of the retina, Studnička for example; but the latter's faith in the proammocoete situation as proving that the lens as a functionless mass antedated the retina is vulnerable to the suggestion that a simple asynchrony of lens and retina development obtains here. No one would suggest that an immature muscle represents a functionless vestige merely because it is present before its proper nerve grows out and reaches it!

If the lens anlage did not lie so neatly in series with organs of indubitable lateral line origin—the olfactory and otic anlages—it is likely that few would ever have thought that the lens antedated the retina, and these few would have abandoned the idea when confronted with the normal dependence of lens formation on the chemotactic influence of the optic cup. To follow Schimkewitsch, however, it seems necessary to believe that the lens antedated the retina and yet was never å lateral line organ, for his theory consumes a pair of such organs to form the lateral retinas but offers these retinas a pair of preformed lenses in the same metamere.

Franz's version is interesting also. He suggested (personal communication) that the lens evolved when the neural tube was just closing or had just closed (fig. 8 *c*), in such a position as to concentrate light on the photosensitive lining of the diencephalon. Its locus of formation then moved laterally (thus escaping involution and coming to lie in the general head ectoderm) to remain in relation to the evolving lateral retina (fig. 8 *c* and *d*), which appropriated the lens since its need for a light concentrator was more important than that of the relict, persistently photosensitive diencephalic ependyma. This idea is, however, equally dependent, with the epibranchial-placode theory, on the demonstrable existence of a self-differentiatory lens anlage; and, moreover, no ontogenetic conditions support it.

The muscle-nerve analogy employed here serves to remind that even if no partially self-determining lens placodes had ever been seen, the placode theory need never die, for it could always be argued that the present dependence of lens formation on the optic cup is secondary. The chemotropic searching out of a muscle by its budding nerve is obviously secondary. One cannot well imagine a phylogenetic stage in which there were anervated muscles and nerves which in the adult reached only part way out from the central nervous system and connected with nothing. Could not the lens have once had an inherent morphogenetic drive which has simply been supplanted by the more convenient, immediate chemotactic stimulus of the optic cup which one sees in

ontogenetic development—a stimulus so strong that it will operate on "foreign" ectoderm and even, as in regeneration of the lens, on the optic cup's own substance? Analogous supplantations are not wanting. Consider the fact that in one species of bird the development of a secondary sexual character may be under "genetic" control while in another the same character may be brought about by hormones.

Obviously, the pros and cons of the placode theory and its variants are much ado about very little. The only other distinct mechanical theory of lens origin, that of Tretjakoff, has been criticized here. Probably it is best to say, with Keibel, that when the optic cup had differentiated and a use for a lens developed, a lentoid thickening of the surface ectoderm appeared—perhaps called into existence even phylogenetically by chemical emanations from the optic cup. No doubt it has often happened in evolution that a new biochemical has had unpredictable morphologic consequences. This would seem to be the simplest possible theory, and the law of parsimony bids that it be accepted.

Miss Sylvia Hagyard drew the illustrations and reduced the diagrams of the many different artists to a common set of conventions for the facilitation of comparison.

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Obituaries

ROBERT SATTLER, M.D.
1855-1939

Dr. Robert Sattler died in Cincinnati on Feb. 20, 1939, in his eighty-fourth year. He was the son of Dr. George Sattler, a native of Hanover, Germany, who came to this country after taking his medical degree in Germany and settled in Cincinnati, where he practiced. The son graduated from the Miami Medical College in Cincinnati in 1875. Dr. E. Williams induced him to take up ophthalmology, and Dr. Sattler came to New York to work with Dr. Herman Knapp as resident surgeon in the New York Ophthalmic and Aural Institute in 1875. After this, he spent two years in postgraduate work in Vienna, Berlin, and in Utrecht, Netherlands. On his return to Cincinnati in 1878 he became a partner of Dr. E. Williams and Dr. S. C. Ayres. In 1890 he founded the Cincinnati Ophthalmic Hospital and acted as professor of ophthalmology in the Miami Medical College, which soon became the medical department of the University of Cincinnati.

Dr. Sattler was a member of many medical societies, including the American Ophthalmological Society, of which he was president in 1914. He was a regular attendant at medical meetings and published many articles in the ARCHIVES, which were usually on clinical, operative and pathologic subjects connected with ophthalmology.

He was a genial, kindly disposed and most industrious man and an important figure in ophthalmology in this country.

ARNOLD KNAPP.

News and Notes

SOCIETY NEWS

European Society of Structive Surgery.—The fourth international congress under the auspices of the European Society of Structive Surgery will be held in Paris, France, Oct. 5 to 7, 1939. The honorary president of the congress is Prof. P. Sebileau, a member of the Academy of Medicine of Paris, and the president is Dr. L. Dufourmental. During the congress the two following subjects will be discussed: (1) treatment of destructions and deformities resulting from scar retraction of the eyelid and (2) treatment of deformities of the jaw.

Other communications, which will be strictly limited to subjects which come within the scope of reconstructive and plastic surgery, will also be presented. Operative clinics on plastic surgery will be held during the congress. A program of artistic entertainment and a banquet will be featured.

Further information concerning the conference, including the rates which are available for the voyage and the stay in Paris, may be obtained by writing to the secretariat of the congress, Maison de Chirurgie, 9 Rue de Turin, Paris 8e.

Oxford Ophthalmological Congress.—The next Oxford Ophthalmological Congress will be held on July 4 to 6, 1940. The master is Percival J. Hay, and the secretary-treasurer, F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

National Society for the Prevention of Blindness.—The annual conference of the National Society for the Prevention of Blindness is planned for Oct. 26 to 28, 1939. Headquarters for the conference will be the Astor Hotel, New York. A complete program, containing the names of speakers and the exact subjects of individual papers, will be printed when plans for the conference are completed and will be sent to any one applying to the society's offices for copies.

UNIVERSITY NEWS

Dr. Glenn G. Gibson has been promoted to be assistant professor of ophthalmology at Temple University School of Medicine, Philadelphia. Drs. Joseph S. Lynch and Lewis Randall Wolf have been promoted to be instructors in ophthalmology.

Course in Ophthalmology.—The establishment of a course in the fundamental sciences in ophthalmology at the Harvard Medical School as a prerequisite to advanced courses has been found of value not only to the practicing ophthalmologist who feels inadequately prepared in basic fundamentals but to the beginner, in that it introduces him to ophthalmology in the proper manner so that he is fully aware of the broad scope of ophthalmology and the futility of inadequate preparation. On the basis of the experience of the past two years, since the fundamental course was introduced, the entire schedule for all courses is arranged so that the course in fundamentals can be followed by clinical courses in proper sequence, giving a full academic year of study. This year the fundamental course is given from October 16 to December 23. It is followed in January by courses in neurophthalmology and in perimetry and ocular muscles, the latter being conducted by Dr. Bielschowsky.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

ORIGIN OF THE VITAMIN C CONTAINED IN THE OCULAR TISSUES. H. H. PODESTA and J. BAUCKE, Arch. f. Ophth. **139**: 720 (Dec.) 1938.

The ocular tissues contain more vitamin C than most other tissues and also more than the blood. This excess has been explained by some authors as being the result of a synthesis of vitamin in the eye and by others as being the result of a secretory process. The authors of the paper under review have determined the vitamin C content in the ciliary body, iris, aqueous humor, lens and vitreous of rats, rabbits, guinea pigs, dogs, sheep, horses and oxen. In the three last-mentioned species the lens contained more vitamin C than any other ocular tissue, whereas in rabbits, dogs and guinea pigs the highest vitamin concentration prevailed in the ciliary body and the iris. The capacity of the explanted lenses to synthesize vitamin C in vitro was strong in cattle and weak or nil in rabbits. In guinea pigs this capacity to synthesize vitamin C depended on the prevailing intraocular vitamin C gradient. If the animals were saturated with vitamin C and the intraocular gradient from the ciliary body to the lens was steep, the ability of the lenses to synthesize the vitamin was relatively weak. Animals fed a vitamin-free diet had a flat intraocular vitamin gradient and a marked synthetic ability of their lenses. Thus it becomes clear that the vitamin C demands of the eye can be and are actually met from two sources, viz., from the blood and by synthesis in the lens. To what extent each source is made use of varies from one species to the other and in the same animal, depending on its age and nutritional state.

P. C. KRONFELD.

Cornea and Sclera

FILARIAL KERATITIS. MARBAIX and APPELMANS, Arch. d'opht. **1**: 978 (Nov.) 1937.

The authors describe a case in which the diagnosis was made only on the finding of microfilarias in sections. The prognosis for ocular onchocercosis of the eye is serious, blindness being due to corneal inflammation and uveitis. There is no medical treatment capable of destroying Filaria, surgical treatment only being possible when nodules are present. In the case reported there were lesions of the conjunctiva and the cornea but no involvement of the iris, ciliary body and choroid. To establish the diagnosis, it is essential to discover microfilarias in the connective tissue either of the skin or the conjunctiva.

S. B. MARLOW.

ULCUS RODENS (MOOREN). J. A. PEYRET, Arch. de oftal. de Buenos Aires 13: 527 (Oct.) 1938.

A case of Mooren's ulcer is reported in a man 35 years of age. The ulcer was of six years' duration and yielded to treatment with an ointment of bismuth tribromphenate after two months.

The literature of the lesion is fully reviewed.

C. E. FINLAY.

INFLUENCE OF AGE ON THE SENSITIVITY OF THE CORNEA. H. ZOBEL, Arch. f. Ophth. 139: 668 (Dec.) 1938.

The sensitivity of the corneas of about 300 persons was determined by testing each cornea at twenty-five points located on three concentric circles with standardized test hairs after the method of von Frey. The results are expressed in averages of the threshold values for each circle. By arranging these values according to the ages of the persons tested, one finds that the corneal sensitivity increases slightly up to the age of 50; from then on it decreases rather rapidly, to reach a minimum in old age. These variations of sensitivity are more pronounced in the periphery than in the center of the cornea. The threshold value for the pain sense of the center of the cornea of 0.3 Gm. per square millimeter, which was first found by von Frey in 1894, was confirmed by the author of the paper under review. In the form of an appendix he records some observations on patients with various local and systemic diseases.

P. C. KRONFELD.

ATTEMPTED KERATOPLASTY ON A BABY WHOSE CORNEAS WERE DESTROYED BY GONORRHEAL OPHTHALMIA. Z. NIŽETIĆ, Klin. Monatsbl. f. Augenh. 101: 32 (July) 1938.

In a girl aged 4 months there was observed extensive destruction of the cornea of both eyes with perforation due to sloughing of the center, through which opening cortical substance and shreds of iris tissue protruded. Rational treatment of the gonorrheal ophthalmia had been delayed by the midwife, who had omitted to use the Credé method of treatment. Nižetić undertook keratoplasty for special reasons, but without much hope. General narcosis was induced with ethyl chloride and chloroform. After bilateral canthoplasty, sutures were placed below and above the limbus, so as to counteract any pressure on the perforated eyeballs. The remaining cornea of each eye was removed with a hand trephine and Graefe's knife, and a layer was formed for the transplant, taken from the eyes of a premature infant delivered in a state of asphyxia. The prolapsing cortical substance and tissues of the iris were removed. Since no other possibility of fixation was offered, the lids were closed by sutures. The transplants became opaque, but the process of sloughing was stopped by this operation. Future developments will be reported later.

K. L. STOLL.

NATURE AND GENESIS OF THE STRIAE IN KERATOCONUS: REPORT OF CASES. M. SCHMIDT, Klin. Monatsbl. f. Augenh. **101**: 36 (July) 1938.

Schmidt analyzes his findings in 4 cases of keratoconus and, after giving a short résumé on previous research by other authors, reports his own conclusions. The striae in keratoconus consist of folds which may be demonstrated in vivo and which may transiently disappear with a temporary increase in the intraocular tension or on exertion of abdominal pressure. The striae are located in the parenchyma adjoining Descemet's membrane; they may occur under especial mechanical conditions in other corneal anomalies. The striae in keratoconus are caused by corrugation; they develop in the stroma overlying the lamina elastica corneae as a result of the contracting force of the membrane. The direction of the striae depends on the meridian in which the strongest tension and the greatest resistance exists against progressing distention; the striae follow the meridian of the strongest curvature. Opacities at the apex are beneficially influenced by the use of contact glasses, the systematic wearing of which may restore vision; they constitute an important orthopedic measure against the progress of this disturbance.

K. L. STOLL.

Experimental Pathology

IMPLANTATION OF TISSUES INTO THE ANTERIOR CHAMBER, WITH PARTICULAR ATTENTION TO OVARIAN TISSUE AND BIOLOGIC REACTION OF PREGNANCY. V. ACCARDI and G. ROSSI, Rassegna ital. d'ottal. **5**: 3 (Jan.-Feb.) 1936.

The authors implanted into the anterior chamber of rabbits' eyes pieces of tissue of the cornea, iris, sclera, lacrimal gland, pancreas, thyroid, thymus, liver, kidneys, spleen, lungs, striated muscles, cardiac muscle, uterus, dura, fascia lata, periosteum, testicles and ovaries.

One hundred and ten transplants were made. It was found that the anterior chamber was a good place for temporary transplantation of tissues but that all the tissues transplanted became newly formed connective tissue.

In only a small percentage of cases did the authors note that the original tissues remained unchanged for a long while, especially the ovarian tissue. This was considered useful in making a diagnosis of pregnancy in animals. A certain change was seen in the ovarian tissue when the urine of pregnant and of nonpregnant women was injected intravenously into the veins of animals. This was not found true with other tissues.

A. PERZIA.

OCULAR CHALCOSIS. C. DUC, Rassegna ital. d'ottal. **5**: 135 (Jan.-Feb.) 1936.

The author studied the action of copper introduced into the anterior chamber and vitreous of 18 rabbits' eyes. In 3 animals the foreign body was spontaneously eliminated. Biomicroscopic and histologic examinations were done on each eye after a period varying between

nineteen and seventy-six days. A similar study was done with controls without a foreign body. It was found that the eyes remain relatively quiet and that there was scarcely any reaction. Cataract was found in only 1 animal, and it resulted from an injury of the anterior lens capsule.

In histologic sections of 3 eyes in which the metal had been present for seventy-five days, the presence of a few rare steel granules was noted on the endothelium of the cornea, in Descement's membrane near the iridocorneal angle. In this same area a large amount of pigmentation was noted which looked like copper granules; similar granules were seen in eyes not treated with copper. These were hard to differentiate from copper granules because of the hematoxin present.

Since these animals do not live long, it would be impossible to watch for the formation of cupric cataract, as this form of cataract develops only after the metal has been in the eye for many years.

A. PERZIA.

EXPERIMENTS ON THE ACTION OF GALACTOSE ON THE LENS OF THE DOG. V. ORTIZ, Arch. de oftal. de Buenos Aires **13**: 467 (Sept.) 1938.

Reference is made to the experimental production in rats by Yudkin, Arnold, Mitchell and Dodge of an increase in the calcium content of the lenses and also to studies on the calcium content in man and other animals in relation to cataract. The author studied experimentally the calcium content of the normal lens of the dog, the action of galactose when introduced into the eye and its action when introduced into the blood. He arrived at the following conclusions:

1. The normal tenor of calcium in the lens of the dog is 0.46 mg. per hundred grams.

2. The normal weight of the lens of the dog is 463 mg., with a + 78.5 deviation.

3. There is no difference between the lenses on the opposite sides of the body.

4. In cataractous lenses of the dog there is some decrease in the calcium content, but the number of observations is too limited to warrant definite conclusions.

5. The injection of galactose into the anterior chamber does not produce significant changes after four hours.

6. The injection of galactose into the blood stream does not produce any change in the calcium contents of the lens.

7. The administration of galactose by mouth does not produce any lesions of the lens.

8. The calcemia after oral administration of galactose oscillates between normal figures.

9. The results of histologic examination of the parathyroid glands of animals after ingestion of galactose were wholly negative.

C. E. FINLAY.

General Diseases

PROPHYLAXIS OF BLINDNESS IN LEPROSY. S. VALLE, Arch. d'opht. 1: 865 (Oct.) 1937.

In cases of leprosy the anterior segment of the eye is chiefly affected, loss of vision resulting from changes secondary to the inflammatory process. The fundus is almost never affected. Involvement of the eye does not occur when the face is free from tubercles, infiltration and anesthesia. It is impossible to avoid ocular manifestations, insidious in leprosy, the development and chronicity of which result in blindness in spite of care in a large number of cases. Chaulmoogra oil, the main treatment for leprosy, usually tends to provoke blindness. From the point of view of prevention the probable modes of invasion of the eye remain to be determined. The author favors the theory that this occurs through the connections of the posterior conjunctival vessels with the anterior ciliary vessels. If this is proved, cauterization of the conjunctiva about the limbus, peritomy and extirpation of tubercles at the limbus must be carried out if blindness is to be prevented.

S. B. MARLOW.

Glaucoma

MICROSURGERY IN CHRONIC SIMPLE GLAUCOMA. O. BARKAN, Am. J. Ophth. 21: 403 (April) 1938.

This article suggests a modification of Barkan's original operation of opening Schlemm's canal, so that a magnification of 20 diameters is obtained instead of 4. This is obtained by using the binocular corneal microscope attached to a head helmet.

W. S. REESE.

LATE TENSIONAL RESULTS OBTAINED BY IRIS INCLUSION IN VARIOUS TYPES OF GLAUCOMA. L. WEEKERS and J. N. FANCHAMPS, Arch. d'opht. 1: 585 (July) 1937.

The authors are of the opinion that the operation described here is the most efficacious of all the operations for glaucoma. They summarize in three tables the data on 22 cases of chronic glaucoma, 13 cases of acute glaucoma and 14 cases of complicated glaucoma. In the group of cases of chronic glaucoma tension of less than 20 occurred in all but 3 over periods up to five years. In the 3 cases the tensions were 20, 23 and 27. In 1 case it was 7, without harmful results. In all but 1 of the acute cases the tension was less than 20, and in this one it was 30. In only 2 of the 14 cases of secondary glaucoma was the tension above 20 (40 and 70). In the entire series the longest period of observation was six years and the shortest twelve months. As a result of these experiences the authors believe that the iris inclusion operation is the operation of choice for glaucoma.

S. B. MARLOW.

THE PATHOGENESIS OF GLAUCOMA, WITH PARTICULAR REFERENCE TO THE ONCOTIC PRESSURE OF THE BLOOD SERUM. A. LECCISI, Ann. di ottal. e clin. ocul. **66:** 501 (July) 1938.

Leccisi attempted to confirm the data of Dieter, who found that the intraocular tension in glaucoma corresponded to the difference between the oncotic, or colloid osmotic, pressure of the blood and the pressure in the intraocular capillaries. The latter he found elevated in glaucoma, especially in the acute type, while the colloid osmotic pressure was reduced in chronic and acute glaucoma, a fact which might account for more rapid formation of aqueous by the process of ultrafiltration. The author employed the osmometer of Krogh, which records the pressure necessary to maintain the level of serum, the serum being separated from physiologic solution of sodium chloride by a collodion filter. The average colloid osmotic pressure of the serum of 5 normal persons was 40 cm. of water. For 5 patients with acute glaucoma, it was 32.5 cm.; for 10 with chronic inflammatory glaucoma, 35.7 cm.; for 8 with chronic simple glaucoma, 37.8 cm. and for 5 with absolute glaucoma, 27 cm. These figures, while higher for normal persons than the figures of Dieter, show in agreement with his findings a lowering of the pressure in glaucoma, especially in the acute and absolute forms. These findings suggested the possibility of lowering the intraocular tension by intravenous injections of a substance which would produce a prolonged increase in colloid osmotic pressure of the blood, and the author has begun a series of experiments with acacia based on this conception.

A bibliography is included.

S. R. GIFFORD.

Hygiene, Sociology, Education and History

SIGHT-SAVING SCHOOLS AND SCHOOLS FOR VISUALLY DEFECTIVE CHILDREN. M. BARTELS, Klin. Monatsbl. f. Augenh. **101:** 161 (Aug.) 1938.

In a detailed and comprehensive paper, Bartels renders an account of the endeavor to further schools for the purpose of saving sight and helping children with visual deficiency. The first man to advance a request for such institutions was an Austrian, Gaheis, in 1802. The author discusses the divers phases of the subject: the prospect of visually defective children in normal schools, the psychologic reactions of these children, the definition of visual deficiency with references to the eligibility of such children for sight-saving schools and the most efficient method of examination for selecting these children. In discussing the divers forms of sight-saving schools, Bartels refers to those organized in England and in the United States. He stresses the necessity of separating the sight-saving schools entirely from schools for normal children and also from schools for the blind. Children with a high degree of myopia should not be admitted to sight-saving schools as long as they present no actual visual deficiency or are not in danger of losing their vision. The main consideration centers on the question of how the visually defective child may be best prepared for some occupation. In the chapter on the instruction at the sight-saving schools and on their equipment, Bartels refers to detail pertaining to lighting,

writing paper, books and size of print and also to optical aids, such as magnifying glasses and telescopic spectacles. Consideration is given the training of the teachers and to the prospective occupation of the pupils. The recommendations of the Sight-Saving-Class-Exchange of England and Scotland are cited in this connection.

Bartels concludes that sight-saving schools are of great importance and that institutions for the blind are far more expensive. It would be advisable, therefore, to give as good care to those afflicted with visual deficiency as is given to the blind. To quote an American author: "Every cent spent by the communities for the sight saving schools will yield a dollar." The drive for sight-saving schools must begin in the large cities. Methods to include the small cities, towns and country districts will be found later. International attention, begun at a meeting in Paris, France, in 1932, may be furthered and expensive experiments prevented through visits by the teachers at foreign sight-saving schools. A central international office has been established at Paris.

K. L. Stoll.

Lacrimal Apparatus

X-Ray Studies of the Nasolacrimal Duct. E. W. Spackman, Am. J. Ophth. **21**: 518 (May) 1938.

Spackman discusses the roentgen examination of the nasolacrimal duct and cites a number of illustrative cases. He gives the following summary:

"These cases have been briefly discussed in an endeavor to show the assistance that it is possible to give the operating surgeon by X-ray examination in the region of the nasolacrimal tract.

"The normal X-ray appearance of the tract has been studied, and the method of procedure and technical points are indicated.

"The examination is recommended in congenital deformities, infectious processes, obstructions, fistulas, and conditions of the surrounding bones and soft tissues.

"This type of examination can be used to great advantage in all cases of suspected pathology. It will be resorted to more generally if it is kept in mind by the clinician as he studies his cases."

W. S. Reese.

Lens

The Lactic Acid of the Lens. R. Weekers, Arch. d'opht. **1**:707 (Aug.) 1937.

The experiments reported were undertaken to determine what happens to the lactic acid which is formed in the lens. Three possibilities are mentioned: combustion, synthesis into glycogen and diffusion. The results of experiments on diffusion are:

1.—(*a*) The concentration of lactic acid in the lens of the calf and ox is considerably above that of the aqueous humor of the same animal. (*b*) The concentration is greater in the young lens than the old. (*c*) The two lenses of the same animal have equal amounts of lactic acid.

2.—(*a*) When the lens is placed in Ringer solution the lactic acid diffuses into the solution and is lowered in the lens. (*b*) Partial dissection of the capsule does not alter the speed of diffusion. (*c*) Renewing the Ringer solution increases the amount of lactic acid diffused. (*d*) The concentration of the lactic acid remains constant during the experiment and diffusion is increased if dextrose is added to the Ringer solution. (*e*) Placed in repeatedly renewed Ringer solution and then in Ringer solution to which dextrose has been added, the lens makes lactic acid.

3.—Lactic acid can be made to pass into the lens if the Ringer solution contains sufficient lactic acid.

S. B. MARLOW.

CLINICAL AND PATHOGENIC PICTURE OF BLACK OR BROWN CATARACT. Y. SHOJI, Arch. d'opht. 1: 1057 (Dec.) 1937.

The author reviews the cases of black or brown cataract which have been reported in Japan and presents reports of 13 personally observed cases, the total number of cataracts being 36 in 26 patients. Eighty per cent of the cases occurred in persons between 51 and 80 years of age. In the majority of the cases vision before operation was quantitative. Enucleation because of tuberculosis was done in 1 case. Twenty-five eyes were operated on for cataract, one expulsive hemorrhage occurring. Cataract operation improved the vision in 24 per cent of the patients, but the vision was always bad. In the majority lesions of the fundus were discovered. In 5 of 6 cases in which histologic examination was done pigment was found in the lens. The author is of the opinion that the coloring of the lens is due to melanin, which does not ordinarily occur in the form of pigment granules.

S. B. MARLOW.

Lids

SPASMODIC RETRACTION OF THE UPPER LID. J. VOISIN, Arch. d'opht. 1: 391 (May) 1937.

The author points out that the changes which can be attributed to the spasmodic retraction of the upper lid are chiefly of two sorts. In the first place, the vertical diameter of the palpebral space is permanently increased and is evident on inspection by the observer. In the second place, it is in the study of the movements of the lid and of the eye that one of the two following changes are discovered, either an asynergy of these movements related to immobility or delay on downward gaze or movements of elevation of the lid. The latter may be an active retraction on downward gaze. In the first group inspection reveals a true Stellwag sign and in the second the "lid lag" of von Graefe or the paradoxic elevation called the pseudo-Graefe sign by Fuchs, who described it in cases of paralysis of the third nerve. The author states that the occurrence of retraction of the upper lid according to the definition of Morax is uncommon. He describes its occurrence in hyperthyroidism, lesions of the sympathetic nervous system, dental con-

ditions, encephalitis, lesions of the cerebral nuclei and syphilis. He points out that it may be due to two causes, either to hypertonia of Müller's muscle or to hypertonia of the levator muscle. He believes that the term spasmodic retraction should be restricted to permanent modification of the palpebral fissure and that one should eliminate the paradoxic elevation of the lid as described by Fuchs.

S. B. MARLOW.

Methods of Examination

A METHOD OF FLICKER PERIMETRY. L. L. MAYER and I. C. SHERMAN, Am. J. Ophth. **21**: 390 (April) 1938.

Mayer and Sherman describe an apparatus to simplify the procedure of flicker perimetry which permits them to obtain reliable readings to the extreme periphery. Charts are presented showing curves of critical frequency on principal meridians and the visual fields of normal persons taken with a frequency of 20, 30 and 40 alternations per second.

W. S. REESE.

ROENTGENOGRAPHIC EXAMINATION OF THE AREA OF THE LACRIMAL SAC AND THE TECHNIC OF THE ENDONASAL OPERATION ON THE SAC: REPORT OF CASES. H. BRUNNER, Klin. Monatsbl. f. Augenh. **100**: 729 (May) 1938.

Brunner reports some impressions gained after performing 32 endonasal operations for removal of the lacrimal sac. The technic, described in detail, follows the routine of West and Kofler, with the exception that the medial wall of an ectatic lacrimal sac is not excised but is divided into four flaps, after the method of O. Mayer; the sac is filled with physiologic solution of sodium chloride prior to this procedure. The author considers the number of personal operations of this type too small for the conception of a definite opinion as to the value of the method. He points out in this connection that some statistics may be misleading because they do not consider the ultimate outcome. Brunner adds endonasal roentgenographic examination to the usual method of examination; this permits the operator to gain a satisfactory view of the anterior portion of the nasal skeleton which cannot be obtained by the usual general exposure of the skull, and it facilitates the control of the size and changes of the operative opening in the bone. This opening begins to grow smaller, in some instances nine months after the operation. Therefore, a large osseous opening is recommended by Brunner, who agrees with Toti in this respect. A number of roentgenograms and accompanying sketches illustrate this procedure and furnish proof for the fact that small osseous openings are responsible for recurrences. The condition of the soft tissues, on the other hand, may lead to the formation of granulation tissues, even in the presence of a large osseous opening. Attention is drawn to Kofler's suggestions, which should be followed. The results of postoperative treatment seem to be improved by the local use of alcohol.

K. L. STOLL.

Neurology

PARADOXIC ELEVATION OF THE UPPER LID ON DOWNWARD GAZE. J. VOISIN, Arch. d'opht. 1: 896 (Oct.) 1937.

The author suggests that spasmodic retraction of the upper lid is a permanent change in the palpebral window and analogous to Stellwag's sign. The present condition is discovered only when the mobility of the lid is investigated. When the patient looks down the upper lid moves upward instead of following the globe. It is a rare condition, not many cases being discoverable in the literature. Some of these are briefly cited. Two theories have been proposed to explain the condition: (1) that it is due to some change in the nuclei and (2) that there is a derangement or switching of the motor impulses. The author suggests an inverse synergy with the possibility of a subnuclear lesion. He believes such a hypothesis capable of explaining both spasmodic retraction and paradoxic elevation.

S. B. MARLOW.

CHIASMIC SYNDROME CAUSED BY A CRANIOPHARYNGIOMA. G. GRANDI. Riv. oto-neuro-oftal. 15: 72 (Jan.-Feb.) 1938.

A 23 year old man presented a chiasmic syndrome; clinical and roentgen examination gave every indication of a suprasellar adenoma being present. Exploratory craniotomy revealed a craniopharyngioma.

Attention is called to the fact that often it is difficult, if not impossible, to tell the nature of an intracranial tumor from clinical findings. Emphasis is placed on the fact that surgical intervention is indicated at the earliest symptoms of a chiasmic lesion, as the results are usually more satisfactory.

F. P. GUIDA.

A CONSIDERATION OF PARALYSIS OF THE ABDUCENS MUSCLE FOLLOWING LUMBAR PUNCTURES. I. BORSOTTI, Riv. oto-neuro-oftal. 15: 205, 1938.

Borsotti describes a case of meningococcic cerebrospinal meningitis in which intraspinal injections of serum were given. Even though the condition of the patient was improving, after the eighth lumbar puncture paralysis of the right sixth nerve was noted.

There are many factors involved, and Borsotti considers each in its turn and comes to the conclusion that in spite of the paralysis repeated spinal punctures are to be considered primarily.

F. P. GUIDA.

CHANGES OF THE OPTIC NERVES CAUSED BY ARTERIOSCLEROSIS, RESEMBLING FOSTER KENNEDY'S SYNDROME: REPORT OF CASES. M. GLEES, Klin. Monatsbl. f. Augenh. 100: 865 (June) 1938.

Choked disk in one eye and atrophy of the optic nerve with a central scotoma in the other eye are the chief symptoms in the Foster Kennedy syndrome. Marchesani described the multiformity of this disease. Glees reports 3 cases in which the same variation of this syndrome was present. Choked disk was present in one eye of each of these 3 patients, but a central scotoma was absent. The cause of the ocular symptoms was evidently arteriosclerosis, contrary to Kennedy's report, who found neoplasms to be the etiologic factor. Glees explains

the pathogenesis as the consequence of pressure of the arteriosclerotic carotid arteries on the optic nerves, coupled with disturbances of the nutrition, i. e., of the circulation. Unilateral or bilateral changes of the nasal halves of the field of vision were observed in each of these 3 cases. The carotid arteries were visible in the roentgenogram of only 1 patient.

K. L. STOLL.

Ocular Muscles

DIVERGENCE INSUFFICIENCY: A CLINICAL STUDY. A. DEH. PRANGEN and F. L. P. KOCH, Am. J. Ophth. 21: 510 (May) 1938.

After presenting the results of a study of 54 cases of esophoria in which an analysis of the findings seemed to indicate a divergence insufficiency, Prangen and Koch give the following summary:

"Divergence insufficiency is a definite clinical entity that causes considerable asthenopia, particularly in near vision. Four cases of divergence paralysis were observed. It would appear clinically that there is probably a cerebral center for active control of the divergence mechanism. In many cases of this series, the incorporation of prisms, base out, in the correcting lenses was most useful. Hyperphoria occurs frequently in association with the esophoria in these cases, and should probably be corrected by the addition of a vertical prism. These eyes seem to show a combination of pathologic states, such as decreased divergence, anisometropia with irritability, vertical imbalance, subnormal accommodation, beginning presbyopia. Such conditions are associated with functional disturbances and an unstable nervous system. With such a combination ocular discomfort is unavoidable. Nonoperative treatment, as here outlined, has been of definite assistance to 50 per cent of the patients reported in this series of cases. Orthoptic training and operation probably should be tried more frequently in selected cases."

W. S. REESE.

QUESTION OF THE BEST AGE FOR OPERATION FOR STRABISMUS. E. DUHAMEL, Bull. Soc. d'opht. de Paris 50: 352 (June) 1938.

Contradictory answers to the question of the preferred age for the operative correction of strabismus have been given by various oculists. Some of them believe that operation is easier to perform in infancy; others believe that binocular single vision is thus favored.

Asmus, Duane and Rönne recommend operation as soon as strabismus is apparent. Jaensch does not hesitate to operate on infants at the age of 1 year. Priestly Smith operates on children 2 years of age. Dimmer and Elschnig operate when the child is between 4 and 6 years of age. Trousseau waits until the child is between 7 and 12 years of age, while Onfray prefers the age of 10. De Lapersonne considers between 10 and 11 to be the most opportune time, and Heimann is partial to 5 years. Barck waits until the age of 12. Wendell Reeber prefers the fourteenth year. Schweiger and Czermak operate between the fourteenth and sixteenth years.

It would seem that operation for squint is done for two reasons. It is done for a purely cosmetic reason, and the vision of the amblyopic eye does not increase; it is also done on the assumption that binocular

single vision may be secured. From an exhaustive study of the question, Duhamel recommends that if a purely cosmetic effect is to be the purpose of operation it is best to defer it until after puberty. For obtaining functional binocularity, he thinks that the sooner operation is accomplished the better.

A bibliography accompanies the article.

L. L. MAYER.

Operations

TENON'S-CAPSULE TRANSPLANTS IN SURGERY OF THE OCULAR MUSCLES, WITH ESPECIAL REFERENCE TO POSTOPERATIVE DEVIATIONS WITH ADHESIONS BETWEEN THE MUSCLES AND THE EYEBALL. C. BERENS, Am. J. Ophth. **21**: 536 (May) 1938.

Berens describes an operation designed to correct secondary deviations with marked limitation of motion in the field of action of a muscle which has been operated on. He gives the following summary:

"1. Tenon's-capsule transplant, which has been performed on three patients, may prove to be efficacious for the surgical correction of postoperative strabismus in which adhesions have formed between the muscles and the sclera.

"2. The operations have not been performed for a sufficient length of time to allow conclusions to be drawn as to the final results.

"3. Experiments will have to be made before the efficacy and exact method by which this operation achieves results may be finally ascertained."

W. S. REESE.

OPTICOCILIARY NEUROTOMY. ROBERTO F. PEREIRA, Arch. de oftal. de Buenos Aires **13**: 1 (Jan.) 1938.

After reviewing the history of opticociliary neurotomy since its first performance in 1867 by von Graefe and detailing the technic of the operation, Pereira states that it is indicated in cases of absolute glaucoma accompanied by pain and in cases of phthisis bulbi, as abolishing the sensitivity of the cornea will permit the patient to wear an artificial eye. He does not consider that it in any way offers protection against sympathetic ophthalmia.

He reports a case of absolute glaucoma associated with high tension and intense pain in which cyclodialysis had brought no relief, opticociliary neurotomy causing the disappearance of all the congestive and painful symptoms and a reduction of the ocular tension to normal.

C. E. FINLAY.

Orbit, Eyeball and Accessory Sinuses

THE FRAGILITY OF THE CAPILLARIES IN OCULAR TUBERCULOSIS. VAN LINT, Arch. d'opht. **1**: 691 (Aug.) 1937.

Van Lint describes his method of determining the fragility of the capillaries. It consists in the maintenance of 35 mm. of mercury pressure by means of a sphygmomanometer for fifteen minutes, 50 mm. for a

second fifteen minutes and 65 mm. for a third fifteen minutes and then counting the number of petechial hemorrhages which appear in the skin. While the fragility of the capillaries is demonstrable in a number of conditions, it is most marked in tuberculosis. The test is negative if no more than ten hemorrhages are counted; it is weakly positive if twenty are found, average if fifty are found and strongly positive if the number is above that. The author's observations showed that in cases in which the diagnosis was firmly established by other methods of examination, the test was moderately or strongly positive, and each time the cause was other than tuberculosis the reaction was negative or weakly positive. The reaction is discussed in relation to treatment.

S. B. MARLOW.

LIPOGRANULOMATOSIS OF THE ORBIT. F. MUSCATELLO, Rassegna ital. d'ottal. 5: 108 (Jan.-Feb.) 1936.

The author reports a case of lipoid necrosis, a trophic condition of the orbital fat, to which he gives the name "lipogranulomatosis."

He has reviewed the literature and states that his case is the second so far reported. The anatomic and histologic pictures are illustrated. The author concludes that the condition may be the result of a predisposition of the adipose tissue to the action of one or more hormones.

A. PERZIA.

The Pupil

POLYCORIA: REPORT OF A CASE. K. LISCH, Klin. Monatsbl. f. Augenh. 100: 896 (June) 1938.

A man, aged 39, presented the following findings during refraction: The sclera of each eye was bluish, and a moderate degree of microcornea existed. In the iridocorneal angle of the right eye a gray glassy membrane was observed which covered the endothelium of the corneal periphery. Almost complete absence of the stroma of the iris gave it a dull brown appearance. The sphincter of the pupil stood out from the exposed pigment layer of the iris due to its lighter color. Two pupils, surrounded by the sphincter, were present. They reacted to light, dilated on instillation of homatropine and contracted on instillation of pilocarpine. An incompletely formed third pupil lay within a cordlike adhesion of the sphincter. A similar condition existed in the left eye, in which the sphincter surrounded three slitlike pupils, the largest being located in the center of the iris. The blood vessels of the iris were scarce, especially in the right eye; the nuclei of the lenses were clouded; the fundi showed no pathologic changes, as far as could be seen, and each eye was of normal size. The condition in this case was bilateral genuine polycoria with extensive hypoplasia of the stroma of the iris and gray deposits at the periphery of the corneal endothelium, resembling the embryotoxon corneae posterius described by Axenfeld. The fibers of the sphincter, derived from the ectoderm, were attached to the mesodermal tissues in the iridocorneal angle of the opposite side, a condition never observed previously. The origin of the polycoria in this case is

attributed to mesodermal disturbances, which caused the ectodermal disorder, which in turn led to the anomalies of the sphincter.

K. L. STOLL.

Physiology

LIGHT ADAPTATION AT THE FOVEA FOR NORMAL EYES. W. D. WRIGHT, Brit. J. Ophth. **23**: 51 (Jan.) 1939.

The author gives the following summary:

"A new model of the subjective photometer is described, in which the effects of adapting the eye to a given intensity, are recorded by matching the test patch viewed in the light adapted right eye against a similar patch seen in the constantly dark adapted left eye. Observations on more than 100 observers have been recorded and are analysed. The data include the intra-ocular factor; that is, the sensitivity of the right eye relative to the left when both eyes are dark-adapted, the adaptation factor, which gives the factor by which the sensitivity of the eye is reduced for the given adaptation intensity (6,000 photons), and the recovery curve, which records the recovery of sensitivity after the adapting light has been removed. Among normal observers, two types of recovery curve have been found, a direct and a delayed recovery, in the latter of which no recovery is recorded for the first few seconds after the removal of the adapting light. The results are discussed in relation to the susceptibility of different observers to glare, and to the photochemical reaction in the retina and to the application of the method for pathological investigations."

The article is illustrated.

W. ZENTMAYER.

Refraction and Accommodation

TRAUMATIC MYOPIA: REPORT OF FOUR CASES. L. MARUCCI, Rassegna ital. d'ottal. **5**: 327 (May-June) 1936.

The author describes 4 cases of traumatic myopia and reviews the literature on this form of ametropia and also on Frenkel's traumatic syndrome of the anterior segment. He states that the myopia in his first case was due to spasm of the ciliary muscle or to hypertension or both and also to subluxation of the lens. In his second and third cases the myopia was produced by a spasm of the ciliary muscle, and in the fourth case the myopia was caused by a tearing of the zonule with subluxation of the lens in the frontal plane. In 1 of the cases the myopia was over 10 diopters; in the first and fourth cases the hypotension later changed to secondary hypertension.

A. PERZIA.

MYOPIA DURING SCHOOL YEARS. E. JAEGER, Klin. Monatsbl. f. Augenh. **101**: 205 (Aug.) 1938.

Jaeger refracted the eyes of 350 girls, aged from 10 to 16, and of 150 boys, aged from 10 to 18 years between 1926 and 1935. The patients were inmates of two boarding schools and were reexamined at intervals for several years. It was the author's purpose to study the percentage

of myopia, its increase and the possible influence exerted by the mode of living and the surrounding conditions. Myopia of more than 0.75 diopter was fully corrected, and glasses of from this strength to 1.5 diopters were worn only during the lessons; those of a high degree, however, were worn constantly. Myopia of over 5.0 diopters was not encountered. Jaeger found that myopia begins at the average age of 12 years and appears never to develop after the fifteenth year of life. The progress of myopia, on the other hand, had not stopped with 18 years in some cases. The largest percentage of progressive myopia was noted at the ages of 11 and 12 years. There was a correlation between age and bodily development and onset of myopia, but no connection could be discovered with the amount of the mental requirements demanded from those pupils.

K. L. STOLL.

Retina and Optic Nerve

LEBER'S DISEASE. J. E. RAAF and H. L. BAIR, Am. J. Ophth. 21: 384 (April) 1938.

Raaf and Bair briefly review the salient features of Leber's disease and give the following summary of their own cases:

"In this series of four cases of Leber's disease in one family, the bitemporal character of the residual field defect in the two older patients and the homonymous position of the scotomas in the patient with the acute form of the disease suggested a lesion in the region of the optic chiasm rather than in the optic nerve proper. However, roentgenologic examination of the sellar region in the last-named case and in one of the former cases yielded no demonstrable evidence of a causative lesion of the pituitary or adjacent structures. There were no other associated signs of pituitary dysfunction in these cases."

W. S. REESE.

SURGICAL THERAPY FOR DETACHMENT OF THE RETINA BY THERMOPUNCTURE AND BY DIATHERMIC COAGULATION. G. F. COSMETTATOS, Arch. d'opht. 1: 579 (July) 1937.

This article is a report on 52 operations for retinal detachment—24 by Gonin's method and 28 by electrocoagulation. Of the 24 cases in which Gonin's method was used, cure resulted in 14 and improvement in 2; there was no improvement in 4, and mediocre results were obtained in 4. Of the 28 cases in which electrocoagulation was used, cure resulted in 16 and improvement in 6; there was no improvement in the other 6. Comparison of these results shows little difference, cure being obtained in 75.7 per cent of all cases. The author does not believe abandonment of thermopuncture in favor of electrocoagulation to be justified. Exact localization, however, is essential for thermopuncture. He favors this procedure in cases in which the tear is small. If the rupture is large, is a disinsertion or is not apparent, electrocoagulation is his method of choice.

S. B. MARLOW.

OBESITY, POLYDACTYLIA AND PIGMENTARY RETINITIS (LAURENCE-BARDET-BIEDL SYNDROME). A. GABRIÉLIDÈS, Arch. d'opht. 1: 887 (Oct.) 1937.

The author describes a typical case of Laurence-Bardet-Biedl syndrome in which complete investigation from the clinical and laboratory standpoint was negative. The theories proposed to explain the pathogenesis of the condition are briefly reviewed. It seems probable that it arises from a developmental disturbance in which the endocrine glands and especially the hypophysis probably play an important part. It remains to be shown that the retinal changes with or without pigment are also the result of pituitary disturbances.

S. B. MARLOW.

TAY-SACHS AMAUROTIC FAMILIAL IDIOCY: REPORT OF CASES. T. WERNCKE, Klin. Monatsbl. f. Augenh. 100: 665 (May) 1938.

The identity of the amaurotic familial idiocy of Tay-Sachs and of other forms of amaurotic familial idiocy, especially Niemann-Pick disease, is still disputed. The author reviews the literature in point and finds reported observations on about 15 eyes removed from patients who succumbed to Tay-Sachs' disease. He adds to these data the pathologic changes observed in eyes removed post mortem from 4 children, aged 1 year and 8 months, 1 year and 1 month, 1 year and 3 months, and 2 years, respectively. The clinical and the pathologicoanatomic condition coincided in these 4 children, who were of Jewish parentage. The disease became manifest in the sixth month of life, and the children died at about the age of 2. All of the children were originally normal; they then ceased to develop, degenerated, lost weight, became apathetic and acquired a staring, idiotic facial expression. Laughing and crying occurred spontaneously in turns, the motions of the extremities acquired a marionette-like character, and death followed as a result of marasmus or intercurrent diseases, chiefly bronchopneumonia. Atrophy of the optic nerve and bluish white discoloration of the macula with a cherry red spot in the fovea were observed in each patient. Histologically, there were observed edema of the retina, preferably of the external layers; edema or exudations between the rods and the cones toward the pigment layer and lamina; distention; formation of vacuoles; degeneration and atrophy of the amacrine cells and absence of Nissl bodies. Werncke points out some differences in his findings from those of other writers and a phenomenon, not previously described, namely, a dark spot on the temporal side of the fovea, observed under transillumination. According to Werncke, who accepts Leber's explanation, this dark spot is caused by edema. The pathogenesis of this spot is discussed in detail.

K. L. STOLL.

PERIARTERIAL SYMPATHECTOMY OF THE COMMON AND INTERNAL CAROTID ARTERIES WITH REMOVAL OF THE CAROTID GLAND: ITS INFLUENCE ON THE DEGENERATIVE PROCESSES IN THE RETINA, AND THE ATROPHY OF THE OPTIC NERVE; REPORT OF CASES. Z. NIŽETIĆ, R. SPIRIDONOVIĆ and S. BUKUROV, Klin. Monatsbl. f. Augenh. 100: 817 (June) 1938.

The authors render an account of the history of sympathectomy and the physiologic considerations leading to its performance. The carotid

gland, or the intercarotid ganglion, is a small sensory organ which may readily be overlooked in its surrounding connective tissue. It is probably destined to register certain qualitative changes of the blood. The authors give a detailed description of their own method of sympathectomy. They used this method in 40 cases, either unilaterally or bilaterally, on the common, or internal, carotid artery, with or without removal of the gland. Of these 63 sympathectomies, 32 were done for atrophy of the optic nerve, 5 for pigmented retinitis, 2 for chorioretinitis caused by hereditary syphilis and 1 for retrobulbar neuritis; 20 cases in which the operation was not successful are not included in this compilation, whereas 20 other cases in which a certain result was obtained are described.

The authors conclude that sympathectomy undoubtedly exerts an evident effect on the circulation of the retinal vessels and on general blood circulation, no matter whether the operation is performed on one or both sides or with or without removal of the carotid gland. It influences, in a certain beneficial degree, the vision and the process of the ocular disease, probably in consequence of improved nutrition. It is difficult to state whether the improvement is permanent. Sympathectomy may be safely combined with decortication of the internal, or common, carotid artery, and its effect remains unchanged if combined with removal of the carotid gland. The effect is increased by bilateral operation, but the result may be transient and waning after some time. The intraocular pressure has to be lowered simultaneously by drugs or operations. Adjuvant therapy is indicated, especially in cases of recent involvement, which offer the best prognosis.

K. L. Stoll.

Tumors

A Cyst of the Posterior Chamber with a Microscopic Study of the Eye. E. F. Krug, Am. J. Ophth. 21: 413 (April) 1938.

Krug cites the case of a boy aged 12 years who sustained a wound extending from the limbus down and out 10 mm. into the sclera. He recovered completely. Nine years later he was seen again because of pain in this eye, and a cyst was found behind the iris in the lower temporal quadrant. Electrocoagulation was performed twice but was unsuccessful, and the eye had to be enucleated. The eye was examined and the following diagnosis made: "1. Staphyloma, intercalary. 2. Epithelial implantation cyst in posterior chamber."

W. S. Reese.

Ganglioneuroschwannospongioblastoma of the Orbital Cavity. J. Montpellier, P. Toulant and J. Foissini, Ann. d'ocul. 176: 18 (Jan.) 1939.

This article is of interest as it concerns a malignant tumor of the orbital nerve, cases of which are extremely rare, and also because two biopsies done at an interval of eight months showed two different types of neoplasm.

The report concerns a young Arab of 8 years who was seen in June 1937 for exophthalmous of the left eye which had been progres-

sing for two months without any functional trouble. The movements of the eye were entirely preserved, the pupillary reflexes were normal, vision was equal to that of the other eye and the fundus was normal. No tumor could be found on palpating the orbit, nor could any enlarged glands be found. General examination gave negative results. An exploratory examination was made of the orbit, when a solid tumor was found adherent to the upper side of the orbit. This was removed in two parts, and for a month the patient received roentgen therapy.

The patient was seen again in February 1938, as the exophthalmos had increased with extreme ocular pain. Owing to the rapidity with which the exophthalmos was increasing, exenteration of the orbit was done with considerable difficulty on March 3. Roentgen treatment was again continued. Up to the time of the report the patient had remained in good health.

A detailed description of the two biopsy specimens is given. The first specimen obtained showed a tumor corresponding in structure to a malignant ganglioneuroma, while the second showed the picture of gliosarcoma. Considerable discussion of this finding follows and a bibliography.

S. H. McKee.

Primary Lymphosarcoma of the Conjunctiva. G. Caocci, Rassegna ital. d'ottal. **5**: 55 (Jan.-Feb.) 1936.

The author reports a case of lymphosarcoma of the conjunctiva of the upper lid. After surgical removal of the growth, a metastatic nodule appeared on the temporal region of the same side several months later. The diagnosis was made by histologic examination. The growth could be confused with other proliferative or neoplastic tumors which do not always present a clean delimination of lymphosarcoma.

The article is illustrated.

A. Perzia.

Dictyoma of the Ciliary Body. P. Satanowsky and E. Cramer, Arch. de oftal. de Buenos Aires **13**: 523 (Oct.) 1938.

The authors refer to the rare cases of dictyoma in the literature. The tumor occurs mostly in persons between the ages of 1 and 8 years and has its origin from the nonpigmented layer of epithelium of the ciliary body, which proliferates outward in chains of longitudinal cells, which roll up on themselves and form cavities. Such tumors differ from glioma in noninvasion of the differentiated retina, in expansion outward and in the nonproduction of metastases. Histologic examination of an enucleated buphthalmic eye with ciliary staphylomas of a girl 6 years of age is reported.

C. E. Finlay.

Metastatic Hypernephroma of the Choroid. Fledelius, Acta ophth. **16**: 527, 1938.

In 1930, following an attack of hematuria, a diagnosis of tumor of the kidney was made in a 48 year old man. The right kidney was removed and the diagnosis of hypernephroma established. Early in 1937 small growths were extirpated from the left mandibular and gluteal

regions. These proved to be metastatic hypernephromas. In the fall of 1937 the sight of the left eye became impaired, and in the course of a few months total detachment of the retina developed. The eye was enucleated, and again a metastatic hypernephroma was found.

It invaded the choroid from a broad base about 1 cm. behind the ciliary body. Only 2 or 3 cases similar to this have been reported.

The article contains two photomicrographs showing the histologic structure of the tumor.

O. P. PERKINS.

Uvea

TERTIARY SYPHILIS OF THE CHOROID (GUMMA). G. FAVALORO, Rassegna ital. d'ottal. 5: 671 (Nov.-Dec.) 1936.

The author reports a case of syphilitic choroiditis. A single large round lesion was present in the choroid of a pregnant syphilitic woman who gave birth to a child with all the signs of congenital syphilis. There was no doubt that her ocular condition was syphilitic, either tertiary syphilis or, according to the history, in the stage between secondary and tertiary syphilis. The author states that such cases are rare and that he has seen only 2. Sometimes these lesions are mistaken for choroiditis due to dyscrasias, toxicity and tuberculosis. He states that these lesions usually appear in the posterior segment of the fundus, that they are benign and that all seem to be of the same size. Histologic sections alone would definitely show whether these lesions are secondary or tertiary.

A. PERZIA.

ATROPHIC CHANGES IN THE IRIS IN INTRACAPSULAR CATARACT EXTRACTION. E. KHURGINA and A. DMITRIEVA, Vestnik oftal. 10: 648, 1937.

In order to study the relation between the capsule and the suspensory ligament a careful slit lamp examination was made of 75 patients with various forms of cataracts (6 of these had heterochromia) who were operated on by the intracapsular method. In 15 patients the capsule ruptured during the operation. The age of the patients varied from 33 to 86 years. In most of the elderly patients atrophic changes were observed in the anterior segment of the eye; in these eyes the capsule was rarely opened, and the postoperative course was more favorable than in the younger patients. This study led the authors to the following conclusions:

1. The rupture of the capsule during the operation depends more on the elasticity of the suspensory ligament than on the thickness of the capsule.

2. The ligament of Zinn is not strongly attached to the ciliary body and lens in eyes with advanced atrophic changes, and the capsule therefore does not tear easily.

3. In eyes in which the atrophy of the iris is slight or absent, as in the young, the separation of the capsule meets a strong resistance in the suspensory ligament, and in these eyes the capsule tears easily and postoperative iritis frequently occurs.

O. SITCHEVSKA.

Therapeutics

COD LIVER OIL FOR LOCAL APPLICATION TO THE EYE. G. POSSENTI, Rassegna ital. d'ottal. **5**: 96 (Jan.-Feb.) 1936.

The author has used a salve containing cod liver oil as a base for the treatment of various ocular diseases, such as asthenic ulcers of the cornea, lymphatic keratitis, trachomatous xerophthalmos, trachoma with or without pannus and squamous ulcerative blepharitis. One hundred patients were treated. The author also experimented on rabbits' eyes inoculated with various organisms.

A salve containing 50 per cent cod liver oil acted as an antiseptic in an experimental suppurative condition of the cornea. The good results obtained from the use of the oil were attributed to its bactericidal power and its high vitamin content.

A. PERZIA.

GENERAL TREATMENT OF OCULAR TUBERCULOSIS. G. STROMBURG, Klin. Monatsbl. f. Augenh. **100**: 11 (Jan.) 1938.

Hypersensitiveness to living tubercle bacilli and their toxins and also to other nonspecific albumins is a phenomenon in tuberculous persons that is closely related to the degree of their immunity. Contrary to antitoxic and bacteriolytic immunity, formation of antibodies is not the essential procedure in tuberculosis. The process of immunization is chiefly connected with the cells of the focus and assisted by the blood-forming organs, the lymphatic system and the connective tissue. It is the therapeutic aim in every form of tuberculosis to further this process in a manner which hastens fibrous formations. Exact local treatment in ocular tuberculosis may retard complications, but symptomatic therapy is essential in an attempt to cure any other tuberculous changes which may be hidden in the body.

Two methods of general treatment are available. The first is the specific application of tuberculin and gold preparations. They do not yield any satisfactory results. Tuberculin can produce an immunity only to tuberculin. Its use in 100 of Stromburg's 167 patients was disappointing for the reasons mentioned in the paper. Four patients were treated unsuccessfully with gold preparations. The second method is the nonspecific treatment with climatic and dietetic agents, applied in the mountains. Satisfactory results were obtained with this method at the Höchenschwand Sanatorium in the Black Forest, where 520 patients with ocular tuberculosis were treated. Stromburg contributes these results to the invigorating and stimulating effect of this second method on the general organism. Increased resistive power was the main factor, and this could not be obtained in the lowlands. The strong ultraviolet rays in the mountain climate play an important role; they may be dosed, so as to avoid an overdose in some patients. The same precaution is taken in exposing the patient directly to the sun. Rest cures, gentle exercise and in some instances, forced, but slight, exercise, are valuable adjuvants. Fresh air and nourishing food are paramount requisites.

K. L. STOLL.

Society Transactions

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COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

ALEXANDER G. FEWELL, M.D., *Chairman*

March 16, 1939

WARREN S. REESE, M.D., *Clerk*

RECOVERY OF NORMAL VISION AFTER RUPTURE OF THE CORNEA. DR. MARY BUCHANAN.

The patient, a 19 year old Negress, had been in an automobile accident and sustained a transverse wound of the left cornea from 8 to 2 o'clock through Descemet's membrane, the center of the cornea escaping. The temporal extremity of the wound contained some prolapsed iris. The rest of the iris had disappeared. There was blood in the anterior chamber, and deep ciliary injection was present. The tension was minus.

Treatment consisted of rest in bed, continuous use of cold compresses and instillation of 2 drops of a 1 per cent solution of atropine sulfate and 2 drops of a 1 per cent solution of phenacaine three times a day after the eye was flushed with a 2 per cent solution of boric acid. The eye healed by first intention, and vision of 6/7.5 with correction was obtained.

DISCUSSION

DR. H. MAXWELL LANGDON: I should like to report a case I saw about six or seven years ago. A young man who was driving a car turned the corner and ran into a parked milk wagon. The tailboard was down and loaded with milk bottles, and the front of the car was completely broken in by the collision with the edge of the tailboard. I removed four pieces of glass from the man's lids. The left cornea was cut across the middle and into the sclera about 1 mm. on each side. There was no prolapse of the iris, and the anterior chamber was completely emptied. I employed a conjunctival flap. Complete recovery ensued, and vision was 6/5. In cases of this kind I am much in favor of the use of the conjunctival flap.

DETERMINATION OF SULFANILAMIDE IN AQUEOUS AND VITREOUS AFTER CONJUNCTIVAL AND ORAL ADMINISTRATION. DR. WILLARD G. MENGEL.

This article will be published in full in a later issue of the ARCHIVES.

EXOPHTHALMOS WITHOUT PULSATION DUE TO ARTERIOVENOUS ANEURYSM: REPORT OF A CASE. DR. STIRLING S. MCNAIR.

This article will be published in full in a later issue of the ARCHIVES.

REMOVAL OF ORBITAL TUMORS. DR. JOHN H. DUNNINGTON.

I find the transconjunctival approach entirely satisfactory for removal of deep-seated growths of the orbit and advocate its use in preference to the resection of an osteoplastic flap. In a series of 49 orbital tumors it was not necessary to resort to the Krönlein method in any instance.

ALEXANDER G. FEWELL, M.D., *Chairman*

April 20, 1939

WARREN S. REESE, M.D., *Clerk*

CHEVALIER TAYLOR AND HIS WORKS. DR. BURTON CHANCE.

The library of the College of Physicians of Philadelphia has received in recent years additions to the collection of Chevalier Taylor's works possessed by the college. This collection offers material for a brief sketch of the professional course of that famous quack oculist who toured all the countries of Europe for fifty years in the eighteenth century, operating on cataracts, treating squinting eyes and dispensing infallible salves and lotions.

From the "Treatise on Cataract and Glaucoma," one learns that Taylor couched cataracts and that occasionally when the lens was dislodged into the anterior chamber during couching he might remove it; yet Taylor cannot be credited with deliberate extraction by Daviel's and by others' procedures. One is justified in rejecting any statements of the "Ophthalmiator to the Pope and to Kings and Princes" that he cured squints by exact tenotomy. Certain competent surgical practitioners who had been invited by Taylor to witness his séances reported his procedures as fraudulent and his accounts of his methods as ineffective.

Taylor was a bombastic charlatan who possessed sound knowledge, yet he was unique among the peripatetic operators in that he was not ignorant, and had he not been an otherwise untruthworthy itinerant his exposition of the physiologic function of the eye and of optics might have been accepted. His works deserve attention; the originals, one may believe, were written by Taylor and were duplicated in many languages by employed translators; the number of his works Taylor recorded as over 50.

Of special note in the college is an atlas in Latin and in German, depicting 243 diseased globes; 1 illustration is believed to be the first drawing of a conical cornea.

Three fine engraved portraits of Chevalier are among the treasures of the college; these show how intelligent and attractive a man he was. He was the last of the tribe which had flourished for over a hundred years. His practice, in so many ways approaching scientific comprehension, led qualified men to investigate ophthalmologic problems hitherto deemed by the regular medical profession as unworthy of their consideration, thereby influencing the dawn of modern scientific ophthalmology.

AN ANALYSIS OF OPERATIVE RESULTS FOR CONCOMITANT CONVERGENT STRABISMUS. DR. GLEN C. GIBSON.

Sixty-six patients were operated on for concomitant convergent strabismus. Bilateral recession of the internal rectus muscles was done on 44 and unilateral recession of the internal rectus muscle combined with resection of the external rectus muscle of the same eye on 22.

The preoperative, operative and postoperative results were compared and analyzed, and the following conclusions were made:

1. Graduated and definite amounts of corrections are indicated in the various degrees of strabismus, in the absence of any good reasons to the contrary.

2. The main good reasons to the contrary are:

(*a*) in youth, the absence of contractures, hypertrophy and atrophy of the muscles; (*b*) unusually excessive or diminished muscular action, as shown by the rotation test; (*c*) the presence of various congenital defects, and (*d*) previous operations on the muscles.

3. There are two main causes for unsatisfactory results:

(*a*) undercorrection and overcorrection and (*b*) failure to select the proper procedure for the given case.

4. No statistical confirmation was available which suggested that the recession-resection technic is more applicable to patients with amblyopic eyes than is the bilateral recession procedure.

5. Bilateral recession is ideally suited to young persons with strabismus of grades 1 and 2 who show by the rotation test bilateral equal excess of the internal muscles associated with fairly normal power of the external muscles.

6. Recession and resection are most applicable to persons who have unilateral preponderance of the abnormalities of the muscles and for those with strabismus of grades 3 and 4. It is the method of choice in most cases.

7. The surgical procedure should be directed toward the muscles which are primarily at fault and in proportion to the amount of the overaction or underaction of the muscles as demonstrated by the rotation test.

DISCUSSION

DR. WARREN S. REESE: Has Dr. Gibson any explanation for the fact that the recession-resection method was much more effective for high degrees of squint than for those of low degree?

DR. WILLIAM ZENTMAYER: Was any attempt made to select the cases for the two types of operation, and what was the interval between the time of operation and the final results of operation as now reported?

DR. H. MAXWELL LANGDON: One point to be considered which was not brought out in Dr. Gibson's paper is the difference between the amount of convergence at distance and at near fixation. This, I think, frequently will help one to decide whether recession as well as an advancement should be done. Recently I operated on a lad of 12 years who had a convergence of 20 degrees at infinity and 35 degrees for near vision. I did an advancement of the external rectus muscle of the right eye and recession of the internal rectus muscle. The result was 6

degrees of exophoria at infinity and orthophoria at 33 cm. The patient already had normal vision in each eye and good stereoscopic vision. Had I not done the recession I am quite sure I should not have gotten the result I did, and it was the difference of convergence between distance and near vision that showed to me that recession was needed.

Another point on which I should like Dr. Gibson's opinion is what the results will be eighteen months or two years from now in amblyopic eyes. My experience with these eyes is that they can be corrected by operation but will not stay straight permanently.

DR. GLEN C. GIBSON: In answer to Dr. Reese, the greater effectiveness of the method for high degrees of squint than for low degrees is probably due to the fact that one attains more correction when both lateral rectus muscles of the same eye are operated on than when only one muscle of each eye is employed. In answer to Dr. Zentmayer, most of the cases were unselected. The operation was delayed as long as possible before the final decision was made. The condition which Dr. Langdon mentioned usually prevails in those cases in which there is a convergence excess and the hypertonus of the internal muscles can also be demonstrated by the rotation tests.

DARK ADAPTATION, HEMERALOPIA AND GLAUCOMA. Dr. JACOB B. FELDMAN.

This article will be published in full in a later issue of the ARCHIVES.

THE CLINICAL VALUE OF THE HORIZONTAL PLANE OF EYES. DR. S. WEIR NEWMAYER.

This is a preliminary report of a study of over 5,000 school children, ages 6 to 16 years. The children were referred for possible ocular defects as a causative factor in preventing efficient reading. Failure to learn to read was due to (1) faulty teaching technic and (2) physical defects, congenital or acquired, of the visual apparatus. This paper did not give detailed findings of ametropia, the pathologic picture of the media of the visual apparatus or heterophoria. The abnormalities of ocular movement in the horizontal plane are here considered as oblique twistings or torsions of one or of both eyeballs from a normal horizontal line, occasioned by abnormal development of the skull and orbits with an asymmetry of the skull. The change of plane causes changes in the center of rotation of the eyes and the center of the optic axis and a resulting failure to provide binocular fusion at reading distances. Some deviations from the normal plane are compensated for by tilting the head and acquiring a suitable plane of vision. Most patients demand suppression of the vision in one eye. Visual confusion and mistakes in recognizing certain letters in print are diagnostic in many cases.

The prevalence of abnormalities among 5,000 poor readers in all grades was 15 per cent. Among 600 children in the first grade with poor reading achievement, it was 40 per cent; among 600 children in the upper grades, fifth and sixth, but not grouped on the basis of reading achievement, it was 37 per cent. The percentage of abnormalities was greater in the Negro race.

DISCUSSION

DR. ALFRED COWAN: Dr. Newmayer's discussion has been interesting from the psychologic standpoint. I should like to know how he arrived at his conclusion regarding the obliquity of the plane—whether he started from the horizontal or if he considered the palpebral fissures as being important to determine the plane of the visual lines. Patients with slanting palpebral fissures do not necessarily have visual axes which conform to the shape of the aperture. I should like to know, also, if the visual acuity of these children was taken into consideration and if their refraction errors were carefully corrected before these tests were made.

DR. S. WEIR NEWMAYER: In answer to Dr. Cowan, the 5,000 children received a complete ocular examination. Full and normal vision in each eye does not assure binocular fusion. The presence of an abnormal horizontal plane, signs of reading deficiency and one's knowledge of physiologic optics should be presumptive evidence that this abnormality was a causative factor.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W. WHITE, M.D., *Chairman*

May 15, 1939

RUDOLF AEBLI, M.D., *Secretary*

PSEUDOGLIOMA DUE TO A CONGENITAL RETINAL FOLD IN THE REGION OF THE ENTRANCE OF THE OPTIC NERVE. DR. J. ARNOLD DEVEER.

The eye of a 3 month old infant, enucleated because glioma retinae was suspected, was studied histologically. The changes found may be summed up as follows: (1) microphthalmos, with an embryonal state of the tissue of the iridocorneal angle and the ciliary body; (2) persistence of the hyaloid artery, its glial sheath and postlental hyaloid tissue; (3) abnormal anastomosis between the definitive vascular system of the retina and the hyaloid vascular system; (5) a congenital fold of the retina due to adhesions of the vitreous with traction by the hyaloid and glial tissue, and (6) posterior lenticonus.

The case is unusual because of the peculiar conelike shape of the fold and because of the clinical simulation of retinoblastoma. It is possible, however, that the postlental tissue alone was visualized by the clinicians.

TANGENT SCREEN RULER. DR. LOREN GUY.

The measurements and instructions for making a ruler that will give the degrees on a tangent screen at 1, 2 and 3 meters were presented. The ruler is easily made or can be purchased at a reasonable price. Besides being inexpensive, it has several advantages over a permanently marked screen in that it can be used for the several distances and for

a tall or short person and does away with the necessity of marks on the screen that may attract the patient's attention away from the taking of the fields.

CONGENITAL ANOMALY OF THE OPTIC PAPILLA. DR. SIDNEY A. FOX.

Most authors ascribe adventitious tissue of the optic nerve head to mesoblastic rests of the hyaloid vessels and Bergmeister's papilla. They usually appear in the form of small epipapillary tags or thin membranes covering part of the disk.

A 9 year old boy with amblyopia and an internal concomitant squint of the right eye was examined. The left eye was normal. Ophthalmoscopic examination showed postcortical lenticular and vitreous opacities. There were scattered areas of pigmentation in the retina. The papilla was completely covered with a mass of grayish white connective tissue rising to a height of 5 to 6 diopters. The central portion of this mass was invaginated and seemed to join the normal physiologic excavation at the level of the disk. Around the excavation the mass of tissue rose steeply and the retinal vessels arched upward with and were embedded in this tissue. The vessels then swept downward to emerge on the retinal floor, carrying some of this fibrous covering along. Around the papilla there was a white conus-like area which was probably due to congenital absence of retinal pigment.

DISCUSSION

DR. J. ARNOLD DEVEER: It is interesting to see this demonstration of a clinical case of a condition which no doubt is somewhat related to that represented by the specimen that I presented. The pathologist always says that he wishes he had the opportunity of examining sections of the tissue. I have not had extensive experience with this particular type of lesion, and I do not think I can add anything to Dr. Fox's opinion of this case, which he has studied so closely. I should think that proliferation of glial tissue at this site would be the most likely interpretation.

ACUTE EXUDATIVE CHOROIDITIS: TREATMENT WITH VASODILATORS. DR. WALTER F. DUGGAN.

Acute exudative choroiditis is an acute vascular catastrophe characterized by arteriolar spasm and increased capillary permeability with transudation of plasma, white cells and, in cases of severe involvement, erythrocytes. This causes acute anoxia of the tissue. The healed lesion shows one or two obliterated choroidal arterioles with atrophy of the overlying choriocapillaris and retina.

The juxtapapillary type differs from the usual type only in its location in the fundus. It is not a distinct and separate entity.

The same pathologic changes occur in acute retrobulbar neuritis and in acute spastic closure of the central retinal artery. In 1925 R. Foster Moore noted the similarity between acute exudative choroiditis and closure of the retinal artery.

Acute choroiditis has been assumed to be due to focal infection, tuberculosis, bacterial infection and other systemic diseases. With the multiplicity of treatments in vogue, from five weeks to seven months

are necessary for cure, over three months being required in most cases. Untreated lesions seem to resolve and heal as rapidly.

Histamine or a histamine-like body is suggested as a possible etiologic agent. An increased sensitivity of the arterioles (and capillaries) accounts for the preferential location of the acute lesion.

On the assumption that acute exudative choroiditis is due to localized anoxia of the tissue, 5 patients have been treated with only sodium nitrite (0.1 Gm.) intravenously.

CASE 1.—An 18 year old youth had blurred vision for eight days. The vision was 20/30 —. The fundus showed juxtapapillary choroiditis. He was given fourteen daily injections of sodium nitrite. There was improvement in the lesion after four days; vision was 20/20 in nine days, and the fundus was almost normal in fourteen days. The field defect continued to decrease in size for two months.

CASE 2.—A 23 year old woman had loss of sight in the left eye for five days. The vision was 8/200, and there was acute juxtapapillary choroiditis under the inferior temporal vessels. She received twelve daily injections of sodium nitrite. After four days the vision was 20/70 —, and the lesion was smaller. After twenty-one days the vision was 20/40 —, and the choroidal lesion was barely perceptible. Sometime during the next six weeks the vision improved to 20/20.

CASE 3.—A 34 year old man had loss of sight in the left eye for three weeks. The vision was 20/100, the vitreous was cloudy, and there was a fresh patch of acute choroiditis in the lower nasal quadrant of the fundus. Surrounding the patch there were several old pigmented choroidal lesions. The patient received fourteen injections of sodium nitrite. After five days the vision was 20/50 + 1; the vitreous was clearer, and the choroidal lesion was smaller. On the twelfth day the vision was 20/30 —; on the nineteenth day the vision was 20/20 — 2, the vitreous was clear and a faint whitish haze indicated the site of the original lesion. Seven weeks later an area of depigmentation was the only remaining trace of the acute choroiditis.

CASE 4.—A 23 year old Negro had loss of sight in the upper field of the right eye for three days. The vision was 20/40. The media were clear, and there was marked juxtapapillary choroiditis at the lower and outer portion of the disk, with swelling of the lower half of the disk, many hemorrhages and marked edema of the retina below the lesion. Later, he was seen to have also a closure of the inferior retinal arteries. He was given thirteen daily injections of sodium nitrite. Vision improved to 20/20 after six injections, with little or no improvement in the fundus. The lack of any marked improvement was explained by the severe impairment of both the choroidal and the retinal circulation.

CASE 5.—A woman of 50 had blurred vision in the right eye for two weeks. The vision was 20/100; there were heavy opacities of the vitreous, a small old pigmented choroidal lesion several disk diameters below the disk and a large fresh patch of acute choroiditis in the lower periphery of the fundus. After nine intravenous injections of sodium nitrite, the vision was 20/30 —; the vitreous was clearer, and the area of choroiditis was definitely smaller. Five more injections were given, and the patient was then given 30 mg. of diluted erythrite tetranitrate

daily. Three weeks after treatment was started the area of exudate had decreased to at least half its original size and some old pigment, the result of a previous attack of choroiditis, was visible on one side of the lesion.

A Foreign Body (a Piece of Steel) in the Optic Nerve. Dr. Hugh S. McKeown.

The case reported is that of a 30 year old white man whose vision of the left eye was lost because of a penetrating wound of the globe. A foreign body, steel, entered the limbus, penetrated the iris, injuring the lens capsule and vitreous, and then entered the optic nerve through the scleral ring. (A kodachrome picture, a roentgenogram, a drawing of a gross specimen and a pathologic slide of the globe were shown.)

DISCUSSION

Dr. Elbert S. Sherman: A number of years ago I reported a similar case before this section and showed the enucleated eye with the foreign body in situ. In the posterior part of the eye there was a piece of steel about 5 or 6 mm. long; it was sharp at one end and was embedded in the sclera at the margin of the nerve. One or 2 mm. of the foreign body protruded through the posterior wall of the eye.

Angiomatosis Retinae. Dr. Edward P. Ryan (by invitation).

This article will be published later.

Pulsating Exophthalmos in Neurofibromatosis: Report of a Case. Dr. Wendell Hughes.

Since earliest recollection the patient had been aware of multiple small nodules in the skin over his entire body. At the age of 16 many of the nodules grew larger and became hard and elevated. One nodule on the right leg reached the size of an orange. The lesions were not tender and were slightly pigmented, mobile and soft. Some of them were pedunculated up to 2 cm. in diameter. Distribution was segmental, corresponding to the distribution of the sensory nerve. A nodule excised from the patient's left arm in the Brooklyn Jewish Hospital was diagnosed as neurofibroma. The patient's father, who died of cancer of the stomach, is said to have had a similar condition, and one sister has nodules similar to those of the patient.

Other findings included pulsating exophthalmos of the left eye, blood pressure of 120 systolic and 70 diastolic, and impaired hearing in the left ear. Other cranial nerves were normal. All deep reflexes were active and equal. The Wassermann reaction was negative.

Examination in the Medical Center, Sept. 8 to 13, 1932, showed in the right eye vision of 20/20, clear media and a normal disk and in the left eye vision of 20/40 and definite pulsation of the eyeball, the globe projecting forward 5 mm. at each pulsation. Definite changes in the breath sounds were heard over the globe. There were complete paralysis of the lateral rectus muscle and considerable ptosis of the upper lid associated with impairment of the superior rectus and the inferior oblique muscles. The visual fields were normal.

All over the neck, chest, back and abdomen and extending partially over the legs and arms were elevated soft nodular masses, often pedunculated and painless to touch.

The anterior margin of the left orbit was larger than the right. The left antrum was smaller than the right. The ethmoid cells on the left side were compressed. The lateral wall and roof of the left orbit were absent in part. The optic canal on the left side was absent. The floor of the orbit compressed the left antrum and sloped backward at the lower level. The temporal fossa of the left side was convex outward. In the lateral view the roentgenogram showed that the roof of the left orbit was not entirely absent but extended backward for approximately 2 cm. The left side of the body of the sphenoid bone appeared compressed with absence of the anterior clinoid process.

The calvarium was well outlined with tables of normal thickness and was not unusual in shape. Diploic venous channels were exceedingly abundant throughout the calvarium. The pineal body showed a fleck of calcium and appeared to lie in its usual position. The convolutional impressions were not accentuated. The basal angle was normal. The sella turcica was open and measured 10 by 12 mm. It was eroded somewhat on the left lateral side. Petrous ridges were not unusual. The left sphenoid ridge was absent. The right optic canal was normal. There was no sinusitis.

The impression was that the condition was a deformity of the left orbit with absence of the posterior halves of the roof and the lateral wall and the optic canal.

DISCUSSION

DR. CHARLES ALLEN PERERA: I should like to ask the explanation for the bruit. As I remember, when Dr. Wheeler reported this case he mentioned the absence of bruit as a factor in the differential diagnosis in such cases, and so differentiated them from cases of arteriovenous aneurysm.

DR. WENDELL L. HUGHES: When Dr. Wheeler first reported this case he reported it as a case of arteriovenous communication in the leg. I did not show the roentgenogram of the leg, but there was a deformity of the tibia which was supposed to have been due to an arteriovenous communication there, but on subsequent investigation the lesion in the leg was found to be simply due to a cyst and apparently was not connected with an arteriovenous communication. Whether there is an arteriovenous communication due to erosion of the walls of the blood vessels in the cavernous sinus I do not know, but there may be a communication there which may possibly explain the bruit. Otherwise, I do not know what the explanation is.

DR. ISADORE GIVNER: This patient had no bruit, but when the stethescope was placed over the closed eyeballs and the patient took a deep breath there was a marked difference in the breath sound heard on the abnormal eye from that heard on the normal eye.

Since there is a cranio-orbital connection here, a wonderful opportunity exists for studying intracranial pressure; at the present time the electrocardiographic apparatus has been fixed so that the pulsation of the eye and changes produced by varying the intracranial pressure, such as from coughing and deep respiration, as well as changes that occur

after the intravenous injections of caffeine can be charted. Some interesting studies are being obtained. I hope to present the results of these next fall.

DR. PERCY FRIDENBERG: If I remember correctly, the difference between the bruit in neurofibromatosis and that in pulsating exophthalmos is this: The bruit may be heard in cases of either condition, but the patient with neurofibromatosis does not feel or sense the bruit and the patient with pulsating exophthalmos does; in other words, the patient with neurofibromatosis has no subjective sensation. The explanation is simple. In neurofibromatosis there is a mechanically transmitted pulsation and no decided change in the vascular contents of the orbit. In pulsating exophthalmos there is great dilatation of all the veins, and the pulsation is directly communicated by the circulation.

It would be interesting to note the effects of posture on a condition of this sort. There is a marked difference in pulsating exophthalmos when the patient lies supine. If a similar observation could be made in this case one would expect that when the patient is lying flat the pressure on the globe through the defect in the orbital roof would be much less than when the patient is upright. No matter how much more active the circulation in the brain when the patient is lying down and the head is low, the factor of mechanical pressure following gravity tends to make the brain mass, including the frontal lobe, sag backward instead of downward toward the eyeball.

DR. WENDELL L. HUGHES: Dr. Fridenberg brings up an interesting question in regard to posture. There are two factors that might change the pressure on the orbital contents with changes in posture; viz., the weight of the brain pressing on the eye and the amount of blood in the cerebral vessels and sinuses. In the supine position the former would naturally be less, but the latter would probably increase.

Therefore I do not believe the pulsation would be less when the patient is standing than when he is in the supine position. Is that not true Dr. Givner?

DR. ISADORE GIVNER: There is the same amount of ocular pulsation when the patient is in the supine position as when he is erect. However, as the patient in the erect position gradually bends his body forward from the waist, the globe reaches its maximal position forward, due to the brain mass coming forward. At this time it cannot retract against gravity, and the pulsation is practically done away with to the observation of the unaided eye.

INTRAOCULAR FOREIGN BODIES: COMMENTS BASED ON 120 CONSECUTIVE CASES. DR. ELBERT S. SHERMAN.

I have reviewed and tabulated the records of 120 consecutive private cases of intraocular invasions by foreign bodies, including 8 cases of double perforation. The wound of entrance was in the cornea in 69 cases (57.5 per cent), in the limbus in 10 (8.3 per cent) and in the sclera in 41 (34.2 per cent). The foreign body was located in the vitreous or the retina in 82 cases, in the lens in 10, in the anterior chamber, iris or the posterior chamber in 20 and in the orbit in 8.

Of the 71 cases in which a magnetic foreign body was removed from the vitreous, the anterior route was chosen in 44 (62 per cent) and the

diascleral method in 27 (38 per cent). The final results were practically the same with each. Of those persons from whom the foreign body was removed by the anterior route, 52 per cent were industrially blind and 48 per cent had good vision. Of those from whom the foreign body was removed by the posterior route, 48 per cent were industrially blind and 52 per cent had good vision. In 30 per cent of the former and 44 per cent of the latter the final vision was 20/30 to 20/15.

There were 2 patients with detachment of the retina both of whom recovered without operation. In the cases of diascleral removal it was not found that vision later deteriorated or that retinal detachment occurred. In 13 cases in which the foreign body was removed through the sclera and the final vision was 20/15 to 20/70, the average period of observation was four years.

It is important to remove the foreign body promptly in all cases of recent injury, and it is not always advisable to wait for a roentgen examination. In 71 of the cases reported, the foreign body was removed within forty-eight hours after the accident occurred, and in 46 of these it was removed the same day.

Seven cases of siderosis bulbi were observed.

Fourteen eyes were enucleated, and enucleation was advised but not permitted eight times. There was no sympathetic ophthalmia. Enucleation of a quiet eye with a retained foreign body is not advised unless it is blind.

I do not favor either the anterior or the posterior route exclusively for the removal of a foreign body from the vitreous. The choice should be based on the various factors in the individual case. Experience and adequate equipment are important.

DISCUSSION

DR. HUGH S. MCKEOWN: In the case I reported and in all similar cases, the left eye was the injured one. My patient was right handed. I think that it should be interesting to make observations of this nature in such a large number of cases as Dr. Sherman has presented. He reported that 73 patients, or 61 per cent, of his series had penetrating wounds of the globe due to personal activity. I should like to ask Dr. Sherman the percentage of right and left eyes and the number of right-handed and left-handed persons.

DR. ELBERT S. SHERMAN: I cannot answer that, but I can say that in the 120 cases in this group there was no difference in the number of right eyes and left eyes that were injured. There were 60 right eyes and 60 left eyes. Also, in those cases in which the accident was caused by hammering or striking metal, the injuries were almost equally divided between the right and the left eye. I have not gone into the matter of handedness in these cases. In a number of instances I have inquired concerning it but have not obtained enough information to make a definite statement. My impression is that it is not an important factor in determining the number of injuries of the right or left eye.

DR. MORRIS DAVIDSON: In my practice I see the end results of intraocular foreign bodies handled by other physicians, about thirty a year. The results in approximately a third of the cases are bad immediately owing to traumatic cataract and enucleation. In only about a third are

good results obtained. The rest of the cases are potentially favorable, but various factors mar the results. In a few cases the factor is ineffectual attempt at removal, but in a large number of cases it is delayed removal of the foreign body. Among 23 cases of intraocular foreign bodies recently reviewed for a special purpose, I found 6 cases of intraocular foreign body that had been overlooked, with 4 cases of siderosis and 1 case of chalkosis. Some of the patients were first seen by a general practitioner and others by an ophthalmologist.

The question of siderosis was also gone into by Dr. Sherman. I should like to call attention to the fact that siderosis may arise much earlier than in five months. It depends on whether one looks for it early and uses the microscope and slit lamp and studies the lens capsule. If it is looked for early, it is seen at a much earlier date than in five months.

I might add that two thirds of the foreign bodies I saw involved the left eye and one third the right eye.

DR. ELBERT S. SHERMAN: I have great respect for Dr. Davidson's opinion on any subject concerning traumatism to the eyes, and when he assures me that siderosis can develop in twelve days, I believe him. I have no doubt it does occur early in some cases and can be discovered if careful investigation and proper forms of examination are made. However, I have not observed any case in which the characteristic changes in the iris and lens appeared in less than five months after the injury occurred.

Book Reviews

L'oeil et les maladies professionelles (maladies du travail). By Dr. Ch. Coutela. Pp. 612, with 43 illustrations. Paris: Masson & Cie, 1939.

Ever since Homo sapiens became Homo faber he has been subject to occupational mishaps, and Egyptian papyri prove that he wrote about them almost as soon as he could write. The bibliography is enormous, and the industry shown by Coutela in assembling and digesting it is apparent in every page of this monograph.

The task of classification was the first and most difficult problem, and the author has solved it with skill and judgment. The chapter on dust is a revelation. I counted sixty-five headings denoting the types of dust, each type being subdivided several times. A convenient simplification divides the types into (1) inert, (2) active (caustics and similar agents) and (3) primarily inert but subsequently active. The effect of each type of dust on the lids, corneas, conjunctivas and lacrimal passages is considered in detail. The author discusses every foreign body encountered in industry. The familiar stone, marble, sand, emory, coal and the common metals are represented, as are many less familiar foreign bodies of vegetable and animal origin.

Great thoroughness marks the chapter on gases encountered in industry. Even the poison gases of war are discussed.

In the chapter on diseases produced by physical agents, the author describes the effects of exposure to roentgen ray, radium and infra-red emanations. He also discusses fully many rarer occupational diseases, such as those due to variations in atmospheric pressure (divers) and in external temperature (aviators).

The ophthalmologist who has not had a wide industrial experience will find many surprises in the chapter on professional intoxications. For instance, he probably would be unaware that workers in tea, coffee or vanilla may be afflicted with central scotomas, retinal angiospasm or amaurosis, respectively. The more familiar chemicals, such as lead, mercury, copper, arsenic, nitrobenzene, methyl alcohol and dinitrophenol, are fully discussed.

A large chapter, more than one hundred pages, is devoted to occupational diseases due to parasitic and infectious agents. These are largely confined to workers among animals or furs, and include tetanus, tularemia, sporotrichosis and actinomycosis. A further indication of comprehensiveness is given in this chapter, in which trachoma and syphilis are discussed as occupational hazards of physicians!

The author discusses the prevention of each of the hazards listed, but he does not limit himself to specific considerations. He analyzes the role of fatigue in the production of industrial diseases of the eye and even discusses the medicolegal implications of occupations hazardous to the eye. In fact, nothing relevant seems to have been omitted. No ophthalmologist whose work includes industrial disease can afford to be without this book.

G. M. Bruce.

Estrabismo. By Jorge Malbran and Esteban Adrogue. $10, Argentine currency. Pp. 472, with 126 illustrations. Buenos Aires: El Ateneo, 1938.

This book is the most exhaustive and complete treatise on strabismus yet published and is based on personal conceptions and observations with a review and discussion at considerable length of all the views hitherto propounded by different authors.

The first part is purely physiologic and pertains to visual perception and spatial optic sense in single and binocular vision, with a special study of the latter and a discussion of the different conceptions and theories thereon; the physiology of the optic movements, and fusion, with the methods for its investigation.

The second part is devoted to the pathologic process of ocular motility and deals successively with disturbances in the muscular equilibrium of the eyes; manifest squint, with a study of spatial projection in concomitant squint, of binocular vision in this condition with variations of normal and abnormal correspondence, of the visual field in squint and of strabismal amblyopia; the examination of persons with squint with different apparatus; the pathogenesis of concomitant squint with a discussion of Lagleyze and Bielschowsky's views; the clinical varieties of concomitant squint, with extracts of 252 case reports, and the treatment of squint (optical and surgical).

C. E. FINLAY.

Collected Reprints from the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital. Volume V, July 1935-July 1938. Baltimore: Wilmer Ophthalmological Institute of the Johns Hopkins Hospital, 1939.

The reprints of articles published by the staff of the Wilmer Institute from 1935 to 1938 are collected in a bound volume dedicated to the memory of W. H. Wilmer, whose portrait appears as the frontispiece. The sixty-two articles on important subjects were contributed by Drs. Alan C. Woods, Jonas S. Friedenwald, Frank B. Walsh, Earl L. Burky, C. A. Clapp, Louise L. Sloan and many others. These valuable contributions to ophthalmic literature in the United States make an imposing volume, which bears witness to the excellent work which is being done at the Wilmer Institute. The staff and the able director are to be congratulated.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.
Place: Heidelberg. Time: Sept. 4-6, 1939.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

Ophthalmological Society of the United Kingdom

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 4-6, 1940.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

Società Oftalmologica Italiana

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome, Italy.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome, Italy.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: New York. Time: June 10-14, 1940.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Ophthalmological Society

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. O. Ebert, 104 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Edward Clark, 1305–14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Dake Biddle, 123 S. Stone Ave., Tucson, Ariz.
Secretary: Dr. M. P. Spearman, 1001 First National Bank Bldg., El Paso, Texas.
Place: El Paso, Texas. Time: Nov. 9-11, 1939.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Virgil Payne, Pine Bluff.
Secretary-Treasurer: Dr. Raymond C. Cook, 1005 Donaghey Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Melville Black, 424 Metropolitan Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Shirley H. Baron, 309 State St., New London.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Grady E. Clay, 384 Peachtree St. N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. D. C. Montgomery, 301 Washington Ave., Greenville, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. James S. Shipman, 542 Cooper St., Camden.
Secretary: Dr. Wright McMillan, 23 Passaic Ave., Passaic.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. F. C. Smith, 106 W. 7th St., Charlotte.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Place: Statesville: Time: Sept. 21, 1939.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
Secretary: Dr. Dan Brannin, 1719 Pacific Ave., Dallas.
Place: Houston. Time: December 1939.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd. S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert J. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to June.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St. N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St. W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Arthur M. Yudkin, New York.
Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Adolph Krebs, 509 Liberty Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

St. Louis Ophthalmic Society

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Ernest Sheppard, 927 Farragut Sq. N. W., Washington, D. C.
Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.